Final report

OWMC
Ontario
Waste Management
Corporation

A provincial crown corporation.

ENVIRON
Counsel in Health and Environmental Science

SITE SELECTION PROCESS. PHASE 4A:

SELECTION OF A PREFERRED SITE(S)

Generic Risk Assessment

Volume 2:

Technical Appendices

Prepared for

Ontario Waste Management Corporation

Prepared by

ENVIRON Corporation
Washington, D.C. and Princeton, N.J.
U.S.A.

April 21, 1986

ENVIRON Corporation
Counsel in Health and Environmental Science

April 21, 1986

Ontario Waste Management Corporation
2 Bloor Street West, 11th floor
Toronto, Ontario
CANADA M4W 3E2

Generic Risk Assessment
Volume 2 Report

Dear Sirs:

We are pleased to submit Volume 2 of the Generic Risk Assessment which consists of appendices of supporting technical information to the previously published Volume 1. It should be noted that the quantitative estimates of public health risks provided in this report are not final. The generic risk assessment will be expanded in Phase 4B to include specific information on the selected site and refinements to the methodology used to characterize human exposure.

Very truly yours,

Joseph Highland
Principal

JH/sjr

Enclosures

The Flour Mill, 1000 Potomac St., N.W.
Washington, D.C. 20007 • (202) 337-7444

C O N T E N T S

	Page
INTRODUCTION	iii
APPENDIX A - TOXICITY PROFILES FOR SURROGATE CHEMICALS	A-1
APPENDIX B - SELECTION OF SURROGATE CHEMICALS	B-1
APPENDIX C - ESTIMATION OF ENVIRONMENTAL RELEASES	C-1
APPENDIX D - DERIVATION OF ENVIRONMENTAL TRANSPORT DATA	D-1
APPENDIX E - RELATIVE RISKS FOR TRANSPORTATION OF SPECIAL WASTES, REAGENTS AND ADDITIVES, AND INTER-SITE TRANSFERS	E-1

INTRODUCTION

The following appendices provide supporting technical information to the Generic Risk Assessment documented in Volume I. The content of each appendix is summarized briefly below.

Appendix A - Toxicity Profiles for Surrogate Chemicals describes the methodology used to develop measures of toxicity for the surrogate chemicals used in the risk assessment. The purpose of the toxicity profiles was to determine acceptable or safe levels of exposure associated with the OWMC facility emissions. Profiles are presented for each of the surrogate chemicals documenting the known literature on toxic effects and concluding with the following data outputs:

- Unit Cancer Risks (UCRs) or Acceptable Daily Intakes (ADIs) for chronic exposures
- Acute Safe Exposure Levels (ASELs) and Short Term Public Limits (STPLs) for acute exposures.

Appendix B - Selection of Surrogate Chemicals describes the procedure used to select surrogate chemicals and estimate their concentrations in the organic waste streams to be managed by the facility. The selection process was

designed to represent each category of wastes to be managed at the OWMC facility by the most hazardous constituent of the waste streams within that category. The appendix describes the ranking schemes used to select surrogates for the following sources of releases:

- Fugitive emissions
- Incinerator emissions
- Truck spill
- Tank farm fire.

The selected surrogates and the concentrations are presented, and the limitations of the selection procedure are described.

Appendix C - Estimation of Environmental Releases provides the basis for the estimation of emissions of gaseous pollutants associated with the facility under four possible scenarios:

- Normal facility operating conditions
- Facility upset conditions
- On-site accidents
- Off-site transportation accidents

The estimation methodology is presented for each of these four scenarios with the emphasis primarily on volatile organic chemicals. Emission estimates are included for each of the surrogate chemicals selected in Appendix B at facility capacities of 150 000 t/a and 300 000 t/a.

Appendix D - Derivation of Environmental Transport Parameters provides the supporting details for the models used to predict indirect exposures via ingestion of food and water. Atmospheric deposition rates are provided for each of the surrogate chemicals by both wet and dry deposition processes, and sub-models are described for the following phenomena:

- Leaf surface deposition
- Uptake from soil
- Retention in cattle tissues and milk

The output from the appendix is a summary of surrogate chemical concentrations predicted in leafy vegetables, cattle tissues and milk.

Appendix E - Relative Risks for Transportation of Special Wastes, Reagents and Additives, and Inter-site Transfers provides an assessment of the relative risks posed by a spill on the local or regional access route to the facility for the following commodities:

- Special wastes transported to the facility
- Reagents and additives transported to the facility
- Treatment residues transferred between units in the event that the components of the facility are located at more than one site.

The risks are compared in terms of both the consequence or severity of the commodity being spilled and the potential frequency of the event occurring; and a set of relative hazard indices are presented.

APPENDIX A

TOXICITY PROFILES FOR SURROGATE CHEMICALS

C O N T E N T S

		Page
A-1	METHODOLOGY	
	Chronic Acceptable Daily Intake	A-1
	Unit Cancer Risk	A-5
	Acute Safe Exposure Limit	A-10
	Short Term Public Exposure Limit	A-12
	Absorption Data	A-15
A-2	TOXICITY PROFILES FOR ORGANIC COMPOUNDS	
	Acrylonitrile	A-19
	Benzene	A-21
	Chloroform	A-24
	Cresol	A-26
	Dioxins/Furans	A-28
	Ethanol	A-33
	Formaldehyde	A-35
	Hydrazine	A-38
	Methyl ethyl ketone	A-40
	Methylene chloride	A-42
	Petroleum distillates	A-52
	Phenol	A-56
	PCBs	A-57
	Pyridine	A-59
	Styrene	A-60
	Tetrachloroethylene	A-62
	Tetraethyl lead	A-66
	Toluene	A-67
	1,1,1-Trichloroethane	A-68
	Trichloroethylene	A-71
A-3	TOXICITY PROFILES FOR METALS	
	Arsenic	A-75
	Beryllium	A-78
	Cadmium	A-80
	Chromium (VI)	A-83
	Copper	A-85
	Lead	A-87
	Mercury	A-90
	Nickel	A-92
	Zinc	A-95
A-4	TOXICITY PROFILES FOR ACID GASES	
	Hydrogen Chloride	A-97
	Hydrogen Cyanide	A-99
	Hydrogen Fluoride	A-101
	Hydrogen Sulfide	A-103
	Nitrogen Dioxide	A-105
	Sulfur Dioxide	A-107
REFERENCES		A-111

LIST OF TABLES

		Page
A-1	Standard Values for Dose Conversions	A-6
A-2	Acceptable Daily Intake	A-7
A-3	Unit Cancer Risk	A-9
A-4	Short Term Limits	A-11
A-5	Summary of Inhalation Absorption Factors	A-16
A-6	Summary of Oral Absorption Data	A-17
A-7	Human Effects Reported After Short-term Exposure to Various Levels of Methylene Chloride	A-51

A-1 METHODOLOGY

This appendix provides a toxicity profile for each of the surrogate chemicals used in this report. Each profile provides an overview of the most pertinent toxicity data from which acceptable exposure limits were derived. Depending on the toxicological behavior of the compound and the exposure scenarios considered in the risk assessment, four different exposure limits were developed, as follows:

- Chronic acceptable daily intake (ADI)
- Unit cancer risk (UCR)
- Acute safe exposure limit (ASEL)
- Short term public exposure limit (STPL)

The methodology used to derive each of these limits is described in the following sections, which preceed the toxicity profiles. Information is also presented on the absorption of each chemical via the inhalation and oral routes of exposure. Each toxicity profile presents a review of the most relevant human and animal toxicity data, and then cites published ADI, UCR, ASEL, or STPL values. In cases where no published values are available, ENVIRON determined which of the toxicity studies provided the best and most relevant data, and then calculated an ADI, UCR, ASEL, or STPL.

CHRONIC ACCEPTABLE DAILY INTAKE (ADI)

For those chemicals not known to produce carcinogenic effects, the acceptable daily intake (ADI) is used as the measure of chronic toxicity. The use of the ADI as a measure of chronic toxicity has much precedent in toxicological science; ADIs are commonly used as the basis for establishing acceptable levels of human response to pesticide residues, drinking water contaminants, and food additives.

An ADI is defined by EPA as the amount of a toxic agent in mg/kg body weight/day (or in mg/d for a 70 kg person) which is not expected to result in any adverse effects after chronic exposure in the general population of humans, including sensitive subgroups (USEPA, 1980a). Operationally, ADIs are calculated by dividing the "no observed effect" level derived from animal or human toxicity studies by one or more uncertainty factors also called safety factors. In many cases, the only data available reflect "effect" levels, and various means have been developed to use such data for derivation of ADIs (USEPA, 1980a; NAS 1980).

Although exposure at the ADI is not expected to produce adverse health effects, the ADI is nevertheless directly related to the chronic toxic potential of a substance. Moreover, it is a measure that contains several systematic adjustments designed to yield a common measure of toxicity from diverse data sets.

ADIs have been established for a relatively large number of substances by various program offices of EPA, and these ADIs were adopted for the present ranking exercise without modification. The ADIs developed by EPA's

Environmental Criteria and Assessment Office (ECAO) (USEPA 1982 and 1984) and EPA's Office of Pesticide Programs (OPP) were used when available. ADI values reported in the National Academy of Sciences (NAS) series Drinking Water and Health were also used when EPA values were not available (NAS 1977). ADIs reported in units of mg/d (for a 70 kg person) were divided by 70 kg to yield ADIs in units of mg/kg body weight per day.

For substances for which ADI values have not been established by EPA or NAS, ADIs were estimated based on available human or animal toxicity data. The methods used for estimating ADIs are generally consistent with published EPA methods (USEPA 1980a). For chemicals for which toxicity data were extremely limited, however, it was necessary to introduce additional assumptions (which were incorporated as uncertainty factors) to derive the ADI. Details of the methodology used to estimate ADIs are presented below.

Methodology for Estimating ADIs

ADIs are generally calculated by dividing the no-observed-adverse-effect level (NOAEL) derived from animal or human toxicity studies by one or more uncertainty factors using the following formula:

$$ADI = \frac{NOAEL}{\text{Uncertainty Factor}}$$

The usual method of determining a NOAEL is first to identify the toxic response occurring at the lowest dose level in the most sensitive animal species for which test data are available. The highest dose level at which this toxic response is found to be unobservable is then identified; this is

the no-observed-adverse-effect level or NOAEL. Preferably, the NOAEL used to derive the ADI is based on chronic toxicity data; if no chronic toxicity data are available, subchronic data are used with the application of additional uncertainty factors.

Recommended safety factors of 10, 100, 1000 were used to estimate ADIs depending on the available human or animal data (USEPA, 1980; NAS, 1977):

> A safety factor of 10 is used when the NOAEL is based on chronic human data. This factor is meant to account for intraspecies variability.
>
> A safety factor of 100 is used when the NOAEL is based on chronic animal data. This factor is meant to account for both intra- and interspecies variability.
>
> An safety factor of 1000 is used when the NOAEL is based on subchronic animal data. This factor incorporates uncertainty in extrapolating from subchronic to chronic exposures and includes the two previous uncertainty factors.

In cases where a NOAEL could not be identified, the lowest-observed-adverse-effect level (LOAEL) was used to estimate the ADI. In such cases, additional safety factors from 1-10 were used, depending on the severity of the effect observed. The intermediate safety factors were developed on a logarithmic scale (e.g., 3.2 being halfway between 1 and 10). The safety factors generally chosen were two and five representing approximately one-third and two-thirds, respectively, of the distance between one and ten. The safety factor of two was used in the case of relatively mild effects such as biochemical changes and potentially reversible, mild organ changes. The safety factor of five was reserved for cases where the effects were more

severe such as organ necrosis, neurological effects, reproductive dysfunction and teratogenic effects.

In some cases, calculation of ADIs required dose conversion (e.g., from ppm in diet or mg/m^3 in air to estimate values in mg/kg body weight per day). The standard values for body weight, food consumption, water consumption, and air breathed used for dose conversion are listed below in Table A-1.

A list of all the ADI values included in this appendix is presented in Table A-2. Each potentially affected organ of the body is identified in the tabulation.

UNIT CARCINOGENIC RISK FOR CARCINOGENS

For those chemicals identifed as carcinogens, the unit carcinogenic risk (UCR) is used as the measure of carcinogenicity. The unit risk is defined as the upper limit on the lifetime probability that the carcinogen will cause cancer at a dose of 1 (mg/kg body weight)/day. The use of the term "upper limit" means that the actual risk is not likely to exceed the predicted risk, and that the actual risk is probably lower than the predicted risk and may even be zero (USEPA 1980). The unit risk is the slope of the carcinogenicity dose-response curve at low exposure levels, and, because of the way it is derived, it is constant over a wide range of low dose exposures. It is thus one systematic measure of carcinogenic potency and is suitable for ranking the relative potencies of various carcinogens in the low exposure region.

TABLE A-1

STANDARD VALUES FOR DOSE CONVERSIONS[a]

Species	Body Weight[b] (kg)	Food Consumption[c] (g/d)	Water Consumption[d] (mL/d)	Air Breathed[e] (L/min)
Mouse	0.025	3	5	0.03
Hamster	0.125	15	15	0.08
Rat:				
Adult	0.25	15	25	0.1
Weanling	0.05	15	25	0.05
Guinea pig	0.57	30	85	0.2
Rabbit	2	60	330	1
Cat[f]	2	100	--	0.32
Dog	10	250	500	5
Pig	60	2,400	--	--
Monkey	5	250	500	1.5
Human				
Male	70	--	2,000	rest 7 light work 29 heavy work 43
Female	50	--	2,000	rest 5 light work 16 heavy work 24

(a) Daily food and water consumption and respiratory minute volume vary markedly with animal body weight and age. The standard values presented in this table represent a best estimate for "typical" laboratory animals and are within reasonable limits of the values found in the published literature. In most cases, values have been rounded to two significant figures.

(b) Source: NIOSH (1980).

(c) Source: NIOSH (1980).

(d) Sources: Hamsters -- Hoeltge, Inc.; Humans -- USEPA (1980); Spector (1956); all other animals -- NIOSH (1980).

(e) Source: Rabbit -- Spector (1956); all other animals -- Altman and Dittmer (1972).

(f) Source: Lehman (1959).

TABLE A-2

ACCEPTABLE DAILY INTAKE (ADI)

(Inhalation and Oral Routes)

Chemical	ADI (mg/kg/d)	Organ
Organic Compounds		
Cresols	1.1×10^{-1}	CNS
Ethanol	4.9×10^{-2}	liver
Formaldehyde	1.2×10^{-2}	GI
Methyl ethyl ketone	2.0×10^{-2}	reproductive, CNS
Phenol	1.0×10^{-1}	kidney
Pyridine	9.0×10^{-3}	liver
Styrene	9.5×10^{-1}	liver,kidney
Tetraethyl lead	2.4×10^{-7}	liver, CNS, pancreas endocrine
Toluene	4.3×10^{-1}	CNS
Metals		
Arsenic	4.0×10^{-4}*	CNS
Cadmium	7.5×10^{-4}*	blood
Chromium	2.2×10^{-3}*	liver, kidney
Copper	2.9×10^{-2}	liver, blood
Lead	1.5×10^{-3}	blood
Mercury	5.7×10^{-5}	CNS
Nickel	1.5×10^{-2}*	reproductive
Zinc	2.1×10^{-1}	reproductive, blood

* Oral route only

Unit risks have been developed for a substantial number of carcinogens by EPA's Carcinogen Assessment Group (CAG) and these were adopted for this risk assessment (CAG 1983; USEPA 1982). For other substances that have been designated as either human or animal carcinogens by the Department of Health and Human Services (HHS) or the International Agency for Research on Cancer (IARC), a unit risk score was calculated from carcinogenicity data using the one-hit model. Unless a substance has been designated carcinogenic by EPA, HHS, or IARC, it was not treated as such in this report (IARC, all volumes 1970-1984; HHS-Carcinogen Report References).

Unit risks were calculated using the one-hit model, which takes the following form:

$$\text{Unit risk} = q_1 = \frac{1}{D} \ln \frac{(1 - P_D)}{(1 - P_O)}$$

where P_D = tumor incidence in treated group

P_O = tumor incidence in control group

D = dose in treated group.

A list of the UCR values included in this appendix is presented in Table A-3. The unit cancer risk values are expressed in units of (mg/kg body weight per day)$^{-1}$.

TABLE A-3

UNIT CANCER RISK (UCR)

(Inhalation and Oral Route)

	UCR $(mg/kg/d)^{-1}$
Organic Compounds	
Acrylonitrile	2.4×10^{-1}
Benzene	5.2×10^{-2}
Chloroform	7.5×10^{-2}
Dioxins/Furans	1.6×10^{5}
Formaldehyde	1.12×10^{-2}
Hydrazine	3.9
Methylene Chloride	6.3×10^{-4}
PCB	4.3
Tetrachloroethylene	3.5×10^{-2}
Trichloroethylene	1.9×10^{-2}
1,1,1,-Trichloroethane	1.6×10^{-3}
Metals	
Arsenic	12.7*
Beryllium	2.6
Cadmium	6.6*
Chromium (VI)	41*
Nickel	1.2*

* Inhalation route only

ACUTE SAFE EXPOSURE LIMIT (ASEL)

For several chemicals considered in the risk assessment, only acute exposures were predicted. Acute safe exposure limits (ASEL) were calculated in a manner analagous to that used for chronic exposure ADI (see p. 3), with two significant differences.

First, only data reported for short term exposures, as opposed to chronic exposures, were used to derive the ASEL. Second, the ASEL was based on a NOEL rather than a NOAEL. The short term NOEL was divided by the same safety factors as used for deriving a chronic ADI. In most cases, human data were available for the chemicals of interest in this report, so that a safety factor of 10 would be appropriate. In cases where a NOEL could not be identified, the LOEL was used to estimate the ASEL. In each case of acute exposure where the ASEL was estimated from a LOEL, the LOEL was based on mild effects, requiring the use of either no additional safety factor or a factor of 2.

It should be emphasized that the use of a NOEL for acute exposure studies is considered to be highly conservative and that a no observed adverse effect level (NOAEL) is a more commonly used basis for estimating a safe exposure level. The reason for this is the generally accepted premise (NRC 1979) that some acute effects such as reversible, mild irritation, odor, or sensory motor effects are not adverse if experienced for only a short period. However, to be consistent with the conservative assumptions adopted throughout the risk assessment, any effect (no matter how slight) was considered to be undesirable for the purpose of establishing short term safe exposure levels.

A list of ASEL values for the chemicals reviewed in this report is presented in Table A-4. The more conventional use of NOAELs to establish short term public exposure limits (STPLS) is described in the following section.

TABLE A-4

SHORT TERM LIMITS[a]

Chemical	ASEL[b]	STPL
Organic Compounds		
Methylene chloride	2.5 mg/kg	17 mg/kg
Acid Gases		
Hydrogen chloride	2.6 mg/m^3	4 ppm for 10 min (6 mg/m^3)
Hydrogen cyanide	1.0 mg/m^3	-
Hydrogen fluoride	1.3 mg/m^3	-
Hydrogen sulfide	0.7 mg/m^3	-
Nitrogen oxides	0.2 mg/m^3	-
Sulfur dioxide	0.26 mg/m^3	3 ppm for 10 min (7.8 mg/m^3)

[a] All short term limits based on inhalation exposures

[b] Acute Safe Exposure Levels based on NOEL from short term studies

SHORT TERM PUBLIC EXPOSURE LIMITS (STPLs)

Criteria that can be used to establish short-term exposure limits for air pollutants have been adopted by the National Academy of Sciences (1979). NAS defines short-term exposure as a single exposure, usually lasting 60 minutes or less, but never more than 24 hours. They emphasize that cases where repeated short-term exposure may occur should be differentiated from those in which exposures may never be repeated. Results of human studies should be used to establish these levels in most cases. When human data are not available, the results of animal studies should be used.

The approach used by NAS depends on a substance-by-substance evaluation of each chemical. Exposure limits for compounds, for which reliable human and animal data predict little chance of irreversible effects from a single exposure, can be established directly from the human data with no additional safety factor. If the human data are weak, but the animal data strongly suggest irreversible effects are unlikely from a single exposure, then an uncertainty (safety) factor can be applied depending on the quality of the data. When cancer or some other irreversible effects are expected, then several possible approaches can be used. These include:

- Establish an exposure limit based on an appropriate uncertainty factor as would be done for noncarcinogens; or, depending on the nature of the chemical, include specific warnings to particular segments of the population that may be at additional risk, e.g. in the case of pregnant women exposure to a possible teratogen.

- Use quantitative risk assessment to recommend an exposure level that provides a defined level of risk (i.e. 10^{-5}, 10^{-6} etc.).

Using the above criteria, NAS has established three types of short-term exposure limits. These include short-term public limits (STPLS), short-term public emergency limits (SPELs), and emergency exposure limits (EELs). These have been defined by NAS as follows.

- SHORT-TERM PUBLIC LIMITS (STPLs) are those which relate to predictable single exposures, usually lasting 60 min or less, but never more than 24 h. STPLs are limits that, if not exceeded, are expected to be associated with only a remote possibility of a nonincapacitating, reversible effect in an exposed population. They differ from air-quality standards in that the STPLs are intended for infrequent exposures. A nonincapacitating, reversible effect is one that will not cause a period of disability; will not impair one's vision, visibility, judgment, or ability to breath; and will not interfere with one's ability to escape from the site of the exposure.

- SHORT-TERM PUBLIC EMERGENCY LIMITS (SPELs) are those which relate to unpredictable single exposures, usually lasting 60 min or less, but never more than 24 h, and whose occurrence is expected to be rare in the lifetime of any one individual. SPELs reflect an acceptance of the statistical likelihood of the occurrence of a nonincapacitating, reversible effect in an exposed population. They are designed to avoid significant decrements in performance during emergencies and

might contain no uncertainty factor. The acceptable level of risk will depend on the specific compound in question and the type of effect produced by the compound. SPELs are concentrations acceptable to the public in emergencies, such as civil defense or defense of neighborhood property in the case of fire.

- EMERGENCY EXPOSURE LIMITS (EELs) differ from SPELs only in that they apply to defined occupational groups, such as military or space personnel, rather than the general public. Because the relevant populations at risk are more narrowly defined than the general public and are generally younger and healthier, an EEL for a specific chemical may differ quantitatively from the SPEL for the same substance. These values will depend on the available data and the collective judgment of the committee recommending the exposure limits.

Although NAS recognizes that emergency exposure limits for the general public (SPELs) and emergency limits (EELs) for defined occupational groups, i.e., military personnel, could be different, they currently believe a single set of emergency exposure limits (EELs) are sufficient to protect both populations. NAS realizes, however, that for population groups which have a more narrow range of susceptibility, the exposure limits might be somewhat higher.

For the purposes of this risk assessment, the STPL was adopted as the most suitable criterion for assessing short term exposures. A listing of STPLs for the pertinent compounds in this appendix was presented in Table A-4.

ABSORPTION DATA

An attempt was made to estimate the amount of absorption that might be expected for each of the chemicals reviewed. Approximate estimates of absorption were derived from review of the secondary literature. Where the absorption factor for a particular chemical was given as a range, the value representing the upper bound of the range was adopted.

For each chemical, the exposure pathway on which the ADI and/or UCR was based, was identified, and used in assigning an absorption factor (see tables A-5 and A-6) for each chemical. This absorption factor was then used in the individual risk assessments. Because ADI or UCR estimates derived from animal studies do not adjust for the degree of absorption, an absorption factor of one was adopted for the exposure pathway if this was the same pathway on which the ADI or UCR was based, regardless of the value which was cited in the secondary literature. If the pathway of human exposure is different than the pathway by which the ADI or UCR was derived, then the literature-based absorption factors were used for risk assessments. If little or no quantitative data were available, 100% absorption was assumed. For example, qualitative statements regarding absorption, e.g. "readily absorbed", "rapid uptake", etc. were considered as indicators of 100% absorption. In the case of the acid gases, absorption was considered to be 100% by the inhalation route, because of their high water solubility and small molecular weights. Tables A-5 and A-6 list the absorption assumed for each chemical by the inhalation and oral routes of exposure.

TABLE A-5
SUMMARY OF INHALATION ABSORPTION DATA

CHEMICAL	PERCENT ABSORPTION	SOURCE	ABSORPTION FACTOR
Organic Compounds			
Acrylonitrile	No data		1
Benzene	46.9%	(NAS, 1976)	1
Chloroform	49-70%	(USEPA 1980 [1980])	0.7
Cresol	No data		1
Dioxins/furans	29% (for dioxins adhering to particles)	(Schaum (USEPA) 1984)	0.29
Ethanol	No data		1
Formaldehyde	95%	(Egle, 1972, in USEPA (OTS) 1981)	1
Hydrazine	"Well absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Methyl ethyl ketone	75%	(Tada et al., 1972, in Clayton, Vol. IIC, 1982)	1
Methylene Chloride	"Rapidly absorbed"	(Van Oettingen et al., 1949 in Clayton and Clayton, Vol. II B, 1981).	1
PCBs	Efficient absorption	(Albro and Fishbein, 1972, in USEPA, 1980)	1
Petroleum distillates	(mineral spirits, Stoddard solvent)		1
Phenol	80%	(Piotrowski, 1971, in USEPA 1980)	0.8
Pyridine	"Absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Styrene	66%	(W.H.O., 1983)	0.66
Tetrachloroethylene	57%	(Ogata et al., 1971, in USEPA, 1980)	0.57
Tetraethyl lead	30±10%	(USEPA, 1977)	0.4
Toluene	36-67%	(Carlsson and Lindquist, 1977 in USEPA, 1982)	0.67
1,1,1-Trichloroethane	"Readily absorbed"	(Stewart, 1968, in NTP, 1983)	1
Trichloroethylene	58-70%	(Soucek and Ulachova, 1960, in NIOSH, 1973)	0.7
Metals			
Arsenic	85%	(USEPA 1980)	1
Beryllium	No data		1
Cadmium	50%	(Clayton & Clayton 1981)	1
Chromium VI	5%	(USEPA 1984)	1
Copper	No data		1
Lead	46%	(USEPA 1984)	0.46
Mercury	80%	(USEPA 1980)	0.80
Nickel	50%	(USEPA 1980)	1
Zinc	No data		1

TABLE A-6

SUMMARY OF ORAL ABSORPTION DATA

CHEMICAL	PERCENT ABSORPTION	SOURCE	ABSORPTION FACTOR
Organic Compounds			
Acrylonitrile	95%	(Young et al. 1977, in USEPA, 1983)	1
Benzene	50%	(Health and Welfare, Canada, 1979)	0.5
Chloroform	10%	(Fray, et al. 1972 in USEPA 1980)	1
Cresol	No data		1
Dioxins/Furans	No data		1
Ethanol	"Rapid"	(Casarett and Doull, 1980)	1
Formaldehyde	"Rapid uptake"	(Malorny et al., in USEPA (OTS) 1981)	1
Hydrazine	"Well absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Methyl ethyl ketone	"readily absorbed"	(Clayton & Clayton Vol II. C.)	1
Methylene Chloride	No data		1
PCBs	92-98.9%	(USEPA, 1980)	1
Petroleum distillates	No data		1
Phenol	"Readily and rapidly"	(USEPA, 1980)	1
Pyridine	"Absorbed"	(Clayton and Clayton, Vol. IIA, 1981)	1
Styrene	No data		1
Tetrachloroethylene	"Poorly absorbed"	(Clayton and Clayton, Vol. IIV, 1981)	1
Tetraethyl lead	Adults: 10% Children: 42%	(Kehoe, 1961; Rabinowitz, 1974; Zeigler, et al., undated, in USEPA, 1977)	1
Toluene	"fairly complete"	(USEPA, 1982)	1
1,1,1-Trichloroethane	"Readily absorbed"	(Stewart, 1968, in NTP, 1983)	1
Trichloroethylene	"Virtually complete	(Daniel, 1962, in USEPA, 1984)	1
Metals			
Arsenic	95%	(USEPA 1981)	1
Beryllium	No data		1
Cadmium	10%	(NRC 1977)	1
Chromium VI	50%	(USEPA 1984)	1
Copper	40%	(USEPA 1980)	1
Lead	15%	(USEPA 1984)	1
Mercury	100%	(USEPA 1980)	1
Nickel	10%	(USEPA 1981)	1
Zinc	80%	(USEPA 1981)	1

A-2 TOXICITY PROFILES FOR ORGANIC COMPOUNDS

ACRYLONITRILE

Laboratory investigations show acrylonitrile to be toxic by all routes of exposure, including inhalation, oral administration, and ingestion. In acute and subchronic studies, acrylonitrile produces adverse effects on the lungs, kidney, spleen, central nervous system, cardiovascular system, and hematologic system in studies with rats, rabbits, and guinea pigs. Acrylonitrile was teratogenic when given by gavage to pregnant rats (USEPA 1980a).

Acrylonitrile is carcinogenic to rats by inhalation and ingestion in drinking water. In a two-year study (Quast et al. 1980 as cited in USEPA 1980a), male and female rats were administered 35, 100, or 300 ppm acrylonitrile via the drinking water. An increased incidence of tumors of the ear canal gland (Zymbal's gland), tongue, gastrointestinal tract, brain, central nervous system, and pituitary, mammary, adrenal, and thyroid glands occurred in the exposed rats, with some tumors appearing in the low-dose animals. In a long-term inhalation study, tumors were reported in rats exposed to 20 and 80 ppm, 6 hours per day, 5 days per week (ACGIH 1981). Acrylonitrile was also mutagenic in short-term assays in S. typhimurium and E. coli (USEPA 1980a).

Preliminary epidemiologic evidence suggests that acrylonitrile may cause an increased incidence of cancer of the lung, colon, and lymphatic and hematopoietic systems. However, the results from the studies are confounded by exposures to multiple industrial chemicals (USEPA 1980a; ACGIH 1981). Several studies of Russian and Japanese workers have been reported; toxicity

attributed to occupational exposure to acrylonitrile includes effects on the cardiovascular, hematopoietic, nervous, and immunologic systems and the liver (USEPA 1980a).

EPA's Carcinogen Assessment Group estimates a unit risk for acrylonitrile of 2.4×10^{-1} $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984).

BENZENE

Central nervous system depression is the primary effect of acute benzene exposure in laboratory animals and humans. Inhalation of 2100-4000 ppm benzene by mice, rabbits and rats leads to narcosis and exposure to higher levels of 10 000 ppm or above leads to death often as a result of respiratory paralysis and cardiac dysfunction (Von Oettingen 1940; Leong 1977; Nahum and Hutt 1934; Morvai et al. 1976 as cited in Sandmeyer 1981). The inhalation LC_{50} in the rat has been reported to be 13 700 ppm (Drew and Fats 1974 as cited in Sandmeyer 1981). The oral LD_{50} in the rat ranges from 900 mg/kg for newborns to 4900 mg/kg for old adults (Kimura et al. 1941 as cited in Sandmeyer 1981). Symptoms associated with acute exposures in humans include nausea, giddiness, headache, unconsciousness, convulsions and paralysis (Sandmeyer 1981). Concentrations in excess of 20 000 ppm lead to death if inhaled for 5 or 10 minutes but deaths following more prolonged exposures ranging from 200-1800 ppm have been reported (Von Oettingen 1940 as cited in Sandmeyer 1981).

Chronic exposure to benzene in an occupational setting is causally related to pancytopenia, a reduction in all the major circulating elements in the blood, including erythrocytes, leukocytes, and thrombocytes. In some cases, decreases in only one type of blood element may occur resulting in anemia, leukopenia or thrombocytopenia. Severe cases can progress to aplastic anemia. These types of changes have been reported in settings where benzene vapor ranges around 60-100 ppm (Hardy & Ellis 1948; Wilson 1942 as cited in Cornish 1980).

The most prevalent observation following chronic benzene exposure among laboratory animals is also leukopenia (Cornish 1981). Rats exposed to concentrations around 50-60 ppm for 5-8 h/d for 4-5 d/wk for a total of between 120-756 hours have developed leukopenia (Deichman et al. 1963 as cited in NRC 1980) and, in the case of one experiment, decreased amounts of DNA in white blood cells, decreased myelocytic activity and an increase in relative numbers of red blood cell precursors (Dobushi et al. 1974 as cited in USEPA 1980b). Chronic oral administration of 50-100 mg/kg as well as chronic subcutaneous injection of 800-1320 mg/kg also resulted in leukopenia in rats (Wolf et al. 1956; Braier 1973 as cited in Sandmeyer 1981).

Benzene has occasionally been shown to have toxic effects on reproductive processes and on fetal development. Female rats exposed to 209.7 ppm benzene for 10-15 days prior to mating showed a complete lack of impregnation. The number of offspring among those rats that did become pregnant was inversely proportional to benzene exposure levels in the range from 0.3 to 209.7 ppm (Gofmekler 1968 as cited in USEPA 1980b). No congenital malformations were observed among offspring of mice injected with 3 mg/kg or administered 1 mg/kg by gavage (Nawrot and Staples 1979 as cited in USEPA 1980b). Exposure of 87egnant rats, mice and rabbits to 2200 ppm benzene during the period of organogenesis for 6-7 h/d produced no overtly toxic effects among offspring other than decreased fetal weight and size, variations in the number of sternebrae and ribs and delayed ossification (Green et al. 1977 as cited in USEPA 1980b).

Benzene is recognized as a human carcinogen, based on epidemiological studies and case reports which convincingly demonstrate that exposure to benzene results in an increased risk of developing leukemia. An increased incidence of leukemia has been demonstrated among workers in rotogravure plants and shoe factories in Italy; among Japanese workers exposed to benzene and other chemicals; among rubber industry workers exposed to various solvents including benzene; and among a group of rubber product workers in the United States (Vigliani and Saita 1964; Ishimaru et al. 1971; Andjelkovic et al. 1976, 1977; McMichael et al. 1974, 1975, 1976a,b; Tyroler 1977 all as cited in USEPA 1980b).

Thus far animal studies have not demonstrated a relationship between experimental benzene exposures and leukemia. However, benzene has been associated with other types of cancer in laboratory animals. F344/N rats and B6C3F1 mice administered 0, 50, 100 or 200 mg/kg (male rats) and 0, 25, 50 or 100 mg/kg by gavage (female rats, male and female mice) 5 d/wk for 103 weeks exhibited increased incidence of neoplasms of the following tissues: Zymbal gland, oral cavity, skin, preputial gland, ovary, mammary gland, harderian gland, lung, liver and lymphoid tissue. The incidences of some of these tumors were increased at all dose levels. In addition various non-neoplastic effects on the hematopoietic system, including bone marrow hyperplasia, lymphocytopenia, leukocytopenia and lymphoid depletion were observed (NTP 1984a).

EPA's Carcinogenic Assessment Group has derived a unit risk of 5.2×10^{-2} $(mg/kg/d)^{-1}$ for benzene based on human occupational exposure (CAG 1984).

CHLOROFORM

Chloroform is rapidly absorbed through the lungs, gastrointestinal tract, and to some extent, the skin (Torkelson and Rowe 1981). Oral LD_{50} values in mice, rats and dogs are 0.08, 0.3 and 1.0 mg/kg respectively (Kimura et al. 1971, Hill et al. 1975, Klassen and Plaa 1966, as cited in NRC 1977). High concentrations cause anesthesia and narcosis, gastrointestinal upsets, cardiac effects, and liver and kidney injury (Torkelson and Rowe 1981). A dose of 250 mg/kg caused liver and kidney changes in rats (Torkelson et al. 1976 as cited in Torkelson and Rowe 1981).

Chronically, chloroform most often causes effects in the kidney or liver (Torkelson and Rowe 1981). Liver and kidney injury, characterized by lobular granular degeneration and focal necrosis in the liver and cloudy swelling in the kidneys, was noted in rats, rabbits, and guinea pigs exposed by inhalation to 50 ppm chloroform and in dogs inhaling 25 ppm chloroform 7 hours a day, five days a week for six months (Torkelson et al. 1976, as cited in USEPA 1980c).

Chronic exposure to chloroform vapor ranging up to about 240 ppm was reported to lead to complaints of lassitude, flatulence, dyspepsia, dry mouth, thirst, depression, irritability, and frequent "scalding" micturition among workers. Some workers staggered about in the workplace and experienced nausea after exposure. In another group of pharmaceutical workers, enlarged livers were associated with chloroform exposure.

Increased incidences of delayed ossification of skull bones and wavy ribs were noted in fetuses of rats administered 30 ppm chloroform 7 hours per day on days 6 through 15 of gestation. At 100 ppm, fetuses were reported to have increased incidences of several abnormalities including acaudia, imperforate anus, subcutaneous edema, missing ribs, and delayed ossification of sternebrae (Schwetz 1974, as cited in USEPA 1980c). Doses of 316 mg/kg/d and greater caused acute toxic nephrosis and hepatitis and death of dams, as well as fetotoxicity. In rabbits, oral doses of 100 mg/kg/d or higher were toxic to dams and fetuses (Thompson et al. 1974, as cited in USEPA 1980c).

Chloroform has been shown to produce liver tumors in female mice after oral administration of 128 mg/kg for 78 weeks. Kidney epithelial tumors were observed when an average dose level of 100 mg/kg was administered to rats by gavage for 78 weeks (NCI 1976 as cited in NAS 1977). Roe (1976 as cited in Torkelson and Rowe 1981) noted renal tumors in Swiss mice when administered chloroform at 60 mg/kg/d. However, the significance of these tumors is questionable because they were subsequently seen in control animals of the same strain of mice.

The Carcinogenic Assessment Group of EPA estimates a unit risk for chloroform of 7.5×10^{-2} based on carcinogenicity data from oral studies in animals (CAG 1984).

CRESOL

Exposure to cresol results primarily in adverse effects of the central nervous system, although adverse effects of the liver, lungs, kidneys and spleen have been observed (Deichmann and Keplinger 1982). Cresol may be absorbed through the intact skin or through the mucous membranes of the respiratory and gastro-intestinal tracts. Acute exposure may produce muscular weakness, severe depression, collapse and in severe cases, death. Gastro-intestinal disturbances have also been reported by individuals acutely exposed to cresol. Cresol has a corrosive action on tissues, producing burns and dermatitis. Fatal poisoning may result if substantial areas of the skin are wet by cresol and are not washed immediately after the exposure (H.B. Elkins 1958 cited in ACGIH 1980). Experiments designed to investigate the acute effects of cresol in animals have indicated that exposures must be of high concentration to produce adverse effects. Rats acutely exposed to air saturated with cresol vapors suffered no adverse effects (H.F. Smyth Jr. 1956, cited in W.B. Deichmann and M.L. Keplinger 1982). The following oral LD50s in rats, for the various isomers of cresol have been reported: ortho-cresol 1.35 g/kg, meta-cresol 2.02 g/kg, and para-cresol 1.80 g/kg (W.B. Deichmann and M.L. Keplinger 1963, cited in ACGIH 1980).

Chronic exposure to cresol causes damage to the liver, kidney, and skin. Pathological investigations in humans have shown that the pathological effects caused by chronic exposure to cresol are similar to the effects produced by chronic exposure to phenol. These investigations revealed congestion of the

liver with pallor and necrosis of the hepatic cells, kidney tubule damage (O. Wandel 1907 cited in Deichmann and Keplinger 1982), and lung damage characterized by hyperemia, emphysema, edema, and bronchopneumonia with petechial hemorrhages in the pleura (E. Kathe 1906 cited in Deichmann and Keplinger 1982). In addition, destruction of the cellular cytoplasm of the gastrointestinal tract was observed in animals following oral administration of the compound (Species unspecificed cited in ACGIH 1980).

The USEPA has derived an ADI of 7.9 mg cresol/day (equivalent to 1.1×10^{-1} mg/kg/d for a 70 kg individual), based on a TLV value (USEPA 1984).

DIBENZODIOXINS AND DIBENZOFURANS

The polychlorinated dioxins and furans comprise a large class of chlorinated aromatic hydrocarbons which includes some of the most toxic synthetic organic compounds that have been tested to date. Within this group, the compound 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) has been the most thoroughly studied and is generally considered to be the most toxic. Several of the furan congeners are also known to be highly toxic, but they have not been thoroughly tested.

2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is one of the most toxic substances known. It produces a delayed biological response in many species and is highly toxic at low doses to mammals, including humans. Many human exposures to TCDD result from occupational exposure to 2,4,5-trichlorophenol (2,4,5-TCP) or 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), in which TCDD is a contaminant. TCDD has been identified as the cause of numerous outbreaks of chloracne in humans. In addition, investigators have reported muscular weakness, loss of appetite and weight, sleep disturbances, hypotension, abdominal pain, liver impairment, peripheral neuropathy, psychopathological changes, hyperlipemia and hypercholesterolemia, and porphyria cutanea tarda among exposed persons (USEPA 1980d).

Damage to the liver and thymus are the predominant effects of subchronic administration of TCDD in animals. Degenerative changes in the liver and thymus, porphyria, altered serum enzyme concentrations, and loss of body weight occur in rats fed TCDD (NAS 1977). Young female rhesus monkeys given a

diet containing TCDD for up to nine months showed loss of facial hair and eyelashes, edema, accentuated hair follicles, and dry scaly skin (Allen et al. 1977 as cited in USEPA 1980d). Five of the eight monkeys died from severe pancytopenia.

TCDD is fetotoxic and teratogenic in various animal species. Fetuses from treated rats have shown reduced body weight, a slight increase in intestinal hemorrhage and edema, and an increase in fetal kidney malformations. In addition, a reduction in fetal number and an increase in fetal deaths have been noted. Increased incidences of cleft palate and kidney abnormalities have occurred in the offspring of treated mice (USEPA 1980d). Gastrointestinal hemorrhage has been observed in the fetuses of treated hamsters (NRC 1977).

TCDD is a potent carcinogen. Ingestion by rats of 2.2 ppb or 0.1 μg/kg/d for lifetime induced squamous cancer of the respiratory tract and oral cavity in males and females and liver cancer in females only. In rats fed diets containing 0.001 to 1000 μg TCDD/kg diet, an increased incidence of liver tumors developed in groups receiving TCDD at levels of 0.005 to 5 μg/kg of diet; animals in the higher dose groups died between the second and fourth weeks of treatment (USEPA 1980d).

EPA's Carcinogen Assessment Group (CAG) estimates a unit risk of 1.6×10^{5} $(mg/kg/d)^{-1}$ based on data from oral carcinogenicity studies in animals (CAG 1984). The Ontario Ministry of the Environment has also published an ADI for dioxins of 10 pg/kg which is based on use of a safety factor (OMOE 1985).

We will not use this ADI because it was calculated assuming dioxins are carcinogens for which a threshold exists. At the present time there is not enough data to come to such a conclusion.

The most toxic of the furan compounds are certain polychlorinated dibenzofuran (PCDF) congeners. The toxicity seems to depend on the number and position of the chlorine substituents of a congener and is highest for the tetra-, penta-, and hexachloro- compounds. The most toxic of the PCDFs includes those compounds fully chlorinated at the lateral positions, e.g., 2,3,7,8-tetrachlorodibenzofuran and similar congeners (Buser 1985).

Little is known about the toxic effects of PCDFs in man because there are no recorded cases in which individuals have been exposed to pure mixtures of PCDFs. In the Yusho and Yu-Cheng outbreaks, resulting from widespread ingestion of contaminated rice-oil in Japan and Taiwan respectively, individuals ingested a mixture of polychlorinated biphenyls (PCBs), PCDFs, and polychlorinated quaterphenyls (PCQs). A number of investigators feel that the lingering toxic effects seen in these individuals are primarily due to the TCDF fraction which has been shown to be the most toxic in laboratory studies (Masuda et al. 1985; Miyata et al. 1985; Kashimoto et al. 1985). The authors also support this contention by noting the relatively mild and short-termed symptoms that have been reported in workers exposed to relatively pure mixtures of PCBs. Symptoms most frequently reported in Yusho and Yu-Cheng cases were dermatological effects, including chloracne and pigmentation of nails, skin, and mucous membranes; ocular effects, including eye discharge, swelling of upper eylid, and transient visual disturbance; hepatic enzyme

induction, and a number of subjective complaints including headaches, numbness of the limbs, and weakness (Reggiani and Bruppacher 1985). A number of these symptoms were also seen in babies exposed in utero or as a result of being nursed with contaminated milk.

The toxic effects noted in toxicity studies of PCDF congeners has revealed a toxicity pattern which is almost identical to that resulting from exposure to other halogenated aromatic hydrocarbons, including TCDD. It appears that these compounds act by a common mechanism (Poland et al. 1983).

In a study in which rhesus macaques were dosed with food containing PCB mixtures, TCDD, or 2,3,7,8-tetrachlorodibenzofuran (TCDF), the same clinical toxic manifestations and histopathologic lesions were observed in the animals, although the potencies of the toxicants covered a wide range (McNulty 1985). Monkeys fed 5 μg/kg/d TCDF in the diet showed atrophy of squamous metaplasia of the sebaceous glands, involution of the thymus, and bone marrow hypolasia. The signs of acute toxicity of TCDF occurred at approximately ten times the dose at which these signs were observed for TCDD.

Different animal species appear to differ greatly in their sensitivity to TCDFs. Guinea pigs are the most sensitive of the animals tested, with an oral LD_{50} of 5-10 μg/kg being reported for this species (Moore 1979). Luster et al. (1979) reported on a study in which guinea pigs were given 2,3,7,8-TCDF by gavage at doses of 0.05 , 0.17 , 0.5 or 1.0 μg/kg once weekly for six weeks. At the two highest doses, thymus/body weight ratios were

suppressed and the animals showed suppression of cell-mediated immune function. The authors reported a 30% mortality in the guinea pigs in the highest dosage group.

TCDFs are also potent inducers of the liver microsomal enzyme system. In one study, the ED_{50} (the dose which results in a response in approximately 50% of the population) for the induction of the enzyme aryl hydrocarbon hydroxylase (AHH) in the rat was found to be approximately 0.5 μg/kg/d for five days (Goldstein, 1977).

There is little published information on the reproductive and teratogenic effects of well-characterized PCDFs. It is reported that the treatment of pregnant mice with doses of 10-100μg 2,3,7,8-TCDF/kg on gestation days 10-13 resulted in a dose-related increases in cleft palates and hydronephrotic kidneys in the fetuses (Weber, H. et al. 1984 as cited in Van Den Berg et al., 1985).

No information was identified concerning the carcinogenicity of PCDFs. The structural similarity and parallel toxicity which some of the congeners show with chlorinated dibenzodioxins (i.e., TCDD) gives reason to expect that certain of these (e.g., 2,3,7,8-TCDF) will probably also be identified as potent carcinogens upon testing. For this reason, PCDFs were assumed to be potent carcinogens similar to dioxins and were assigned the unit risk of 1.6×10^{5} $(mg/kg/d)^{-1}$ which, as previously described, was calculated for TCDD by EPA's Carcinogen Assessment Group (CAG 1984).

ETHANOL

The acute toxicity of ethanol is low for both animals and man. The LC_{50} in rats is about 13 000 ppm for 22 hours (ACGIH 1981). The symptoms of intoxication are slight irritation of mucous membranes, ataxia, incoordination, and drowsiness. The narcotic dose for rats is 19 260 ppm for 2 hours (ACGIH 1981). Ethanol is not appreciably irritating to the skin, but has caused mild to severe irritation to rabbit eyes depending on the concentration used in testing (Rowe and McCollister 1982). Concentrations of 250 to 1064 ppm for occupational exposure to ethanol vapor have been suggested as nonirritating to the eye and upper respiratory tract (ACGIH 1981). Teratogenic effects including increased postimplantation death, retardation of fetal development, and minor skeletal variants have been reported in mice and rats (NIOSH 1983).

Prolonged exposure to ethanol has resulted in fatty changes in the myocardium and liver. Rats fed a diet in which 33% of the caloric intake was ethanol for up to 14 weeks developed fatty livers (Rowe and McCollister 1982). Rats, guinea pigs, rabbits and beagle dogs exposed continuously to vapors containing <86 mg/m^3 ethanol for 90 days exhibited no effects as measured by behavior, mortality, hematologic effects, and gross histopathology (Coon et al. 1970 as cited in Rowe and McCollister 1982). Exposure of rabbits to air saturated with alcoholic vapors for 25 to 365 days induced cirrhosis of the liver (Mertens 1896 as cited in Rowe and McCollister 1982). There is no information regarding the carcinogenicity of ethanol.

Based on the 90 day study in rats (Coon el al. 1970 as cited in Rowe and McCollister 1982) and applying safety factors of 100 for extrapolation from animals to sensitive humans and 10 for extrapolation from subchronic to chronic effects, an ADI of 4.9×10^{-2} mg/kg/d is estimated.

FORMALDEHYDE

In humans, adverse health effects may occur after exposure to formaldehyde by inhalation, ingestion, or skin contact. Inhalation of formaldehyde causes eye, skin, and respiratory tract irritation. Effects including eye irritation, lacrimation, ocular damage, itching of the eyes, burning of the eyes and nose, sneezing, coughing, dry and sore throat, pricking irritation of the mucous membranes, upper respiratory tract irritation, primary skin irritation, allergic dermatitis, disturbed sleep, headaches, and unusual thirst developed in workers exposed to formaldehyde vapor (ACGIH 1981). Ingestion of formaldehyde causes headaches, upper gastrointestinal pain, allergic reactions, corrosion of the gastrointestinal and respiratory tracts, systemic damage, and death. Systemic poisoning by ingestion is uncommon because the irritant effects of formaldehyde make ingestion unlikely. Direct contact with aqueous formaldehyde causes skin irritation and ocular damage. Formaldehyde also causes skin reactions in some sensitized people using cosmetic formulations containing only 0.01 percent formaldehyde (NRC 1981).

Formaldehyde is irritating to the eyes, skin, and respiratory tract of laboratory animals. Formaldehyde is not teratogenic in animals by ingestion or inhalation; however, oral doses of 185 mg/kg/d formaldehyde (containing 12-15% methanol as a preservative) on days 6 to 15 of gestation caused death in 22 of 34 pregnant mice (Marks et al. 1978 as cited in NRC 1981). No reproductive effects have been observed to occur in male rats exposed to formaldehyde through drinking water or by inhalation, or in dogs exposed to formaldehyde in the diet. Formaldehyde is mutagenic in several test systems (NRC 1981).

In subchronic inhalation studies in rats, guinea pigs, rabbits, dogs, and monkeys, formaldehyde caused lung, heart, and kidney inflammation; decreased liver weight; eye and respiratory tract inflammation; decreased weight gain; and decreased food consumption.

In a chronic inhalation study in rats and mice, sponsored by Chemical Industry Institute of Toxicology (CIIT), formaldehyde caused squamous cell carcinoma of the nasal cavity in rats exposed to 15 ppm, 6 hours/day, 5 days/week for 18 months. At the Formaldehyde Symposium held in Raleigh, N.C., in November 1980, CIIT reported that nasal cancer occurred in rats exposed to 6 ppm for 24 months and in mice exposed to 15 ppm for 24 months (CIIT 1980, as reported in NRC 1981).

The estimated dose-response curve for formaldehyde is very steep. For this reason, the UCR based on the upper 95% confidence limit derived over a large range of exposure concentrations yields extremely conservative and unreliable risk estimates. The UCR used in this analysis was based on the upper 95% confidence limit of the dose-response curve limited to the range of doses of concern estimated in the exposure scenarios. The UCR calculated in this manner is 1.12×10^{-2} $(mg/kg/d)^{-1}$ and still should be considered to be a very conservative estimate. For example, the risk calculated using the above UCR for a theoretical exposure of 3.2×10^{-5} ppm over 30 years of a 70 year lifetime is 2.6×10^{-7}. In contrast, the maximum likelihood estimate of risk for the same scenario is 8.1×10^{-22}, a difference of 15 orders of magnitude. The UCR based on the 95% upper confidence limit was used in the analysis, despite the discrepancies involved, in order to be consistent with other UCRs used in the analysis.

Because there is no evidence that formaldehyde is carcinogenic by ingestion, a toxicity reference dose was estimated for noncarcinogenic effects of formaldehyde. Dubrenil et al. (1976 as cited in NRC 1981) exposed rats continuously to 1.6 ppm formaldehyde for 45 to 90 days. At this dose level, hair discoloration was evident. At a higher dose level weight gain was reduced. Applying a safety factor of 100 to the no adverse effect dose of 1.15 mg/kg/d yields an estimated ADI of 1.2×10^{-2} mg/kg/d.

HYDRAZINE

Hydrazine is a strong skin and mucous membrane irritant, convulsant, and has hepatotoxic and mild hemolytic properties (Reinhardt and Brittelli 1981). Exposure to high concentrations of the vapor results in eye and respiratory tract irritation, lung congestion, bronchitis and pulmonary edema. In rats, the oral LD_{50} is 60 mg/kg and the inhalation LD_{50} for 4 hour exposure is 570 ppm. (Reinhardt and Brittelli 1981). Hydrazine is rapidly absorbed through intact skin, and also through the lungs and gastrointestinal tract. (IARC 1974a).

Hydrazine has been shown to be carcinogenic in mice and rats after oral administration. The major target organ is the lung, although hepatocarcinomas have also been noted (IARC 1974a). In mice fed 21 mg hydrazine sulfate for up to 78 weeks, the incidence of pulmonary tumors was 54% in males and 32% in females, compared with control incidences of 24% and 4% in males and females, respectively (Biancifiori 1970 as cited in IARC 1974a). In rats administered 18 mg hydrazine by stomach tube for 68 weeks, incidences of pulmonary tumors were 3/14 and 5/18 in males and females, respectively (Severi and Biancifiori 1968 as cited in IARC 1974a). In this study, no tumors were found in the untreated animals.

Although it has not been extensively studied, there is no epidemiological evidence that hydrazine is carcinogenic in humans. Roe (1978 as cited in ACGIH 1981) reported no increase in tumor incidence in workers from a

hydrazine manufacturing plant in operation from 1945 to 1970. The Carcinogen Assessment Group of EPA has derived a unit risk of 3.9 $(mg/kg/d)^{-1}$ for hydrazine based on oral animal studies (CAG 1984).

METHYL ETHYL KETONE

Acute exposure of humans to methyl ethyl ketone causes irritation of the eyes, nose, and throat (K.W. Nelson et al. 1943 cited in W. J. Krasavage et al. 1982). Exposure to higher concentrations of methyl ethyl ketone (300-500 ppm) also produces nausea and headache (H.B. Elkins 1959 cited in Krasavage et al 1982). In addition, methyl ethyl ketone causes irritation and corneal damage when splashed in the eyes (Clayton and Clayton 1982). In animals, acute exposure to methyl ethyl ketone causes irritation of the eyes and nose and may also cause narcosis or death. Exposure of rats to 4000 ppm for 2 hours produced death in 4/6 animals exposed (H.F. Smyth Jr. 1956 cited in Krasavage et al 1982). Exposure of guinea pigs to 3000 ppm, however, produced no observable adverse effects. The following oral LD50s for rats have been reported 3.3, 3.4 and 3.6 g/kg (E.T. Kimura et al 1971 and H.F.Smyth 1954, cited in Krasavage et al 1982).

Workers chronically exposed to methyl ethyl ketone by inhalation or by skin contact developed numbness in their upper extremities (A.R Smyth and N.R. Mayers 1944 cited in Krasavage et al 1982). In addition, prolonged dermal contact may defat the skin and cause dermatitis (K.E. Malten et at 1968 cited in Krasavage et al 1982). Chronic exposure of animals, by various routes of exposure, does not appear to produce adverse central nervous system effects. Cats and dogs receiving subcutaneous injections of methyl ethyl ketone 5 days per week for 8.5 months showed no signs of nervous system damage (P. S. Spencer and H. H. Schaumburg 1976, cited in Krasavage et al. 1982). Rats

receiving intraperitoneal injections of the compound for 35 weeks suffered no neuropathological changes (Unpublished data, Eastman Kodak Company cited in Krasavage et al. 1982). Finally, there was no evidence of neurotoxicity in rats following exposure by inhalation to 4740 ppm for 4 weeks or to 2150 ppm for 6 weeks (P.V. DeJesus et al. 1977, cited in Krasavage et al. 1982).

In a study of the teratogenic effects of methyl ethyl ketone, pregnant rats were exposed to 1000 ppm or 3000 ppm methyl ethyl ketone for 7 hours per day on days 6 through 15 of gestation. No maternal toxicity was observed at either dose level. Litters of rats exposed to 1000 ppm exhibited an increased incidence of skeletal abnormalities; litters exposed to the higher dose level had an increased incidence of sternebral variations, and soft tissue alterations (Schwetz et al. 1974 cited in Krasavage et al. 1982). A repeat of this study indicated that neither dose level produced teratogenic or embryotoxic effects. The slight increased incidence of fetotoxic effects observed in the first study was also noted in the second study (M. M. Deacon et al 1981 cited in Krasavage et al 1982).

The USEPA has derived an ADI of 1.40 mg methyl ethyl ketone per day (equivalent to 2.0×10^{-2} mg/kg/d for a 70kg individual) based on a NOEL obtained in a subchronic inhalation exposure of rats (USEPA 1984).

METHYLENE CHLORIDE

The central nervous system appears to be the primary target organ for methylene chloride. In humans accidental inhalation of high concentrations of methylene chloride of the order of about 20 000 ppm leads to central nervous system (CNS) depression, ultimately resulting in narcosis and perhaps death (Moskowitz and Shapiro 1952 as cited in IPCS 1984). Signs of CNS depression, narcosis, irritation of the eyes and respiratory tract, and lung edema have been reported following exposure to smaller concentrations of methylene chloride vapor via the use of paint removers, and have been associated with corresponding increases in carboxyhemoglobin levels which are indicative of methylene chloride metabolism. Exposure to methylene chloride in a poorly ventilated room has also been associated with the occurrence of myocardial infarction (IPCS 1984).

Exposures of mice to 5000 ppm methylene chloride continuously for 7 days had little permanent effect. Subjects responded to exposure with excitement and increased activity which was followed by decreased activity and "hunched" posture, but by later in the exposure period the mice were virtually normal (Weinstein et al. 1972 as cited in USEPA 1985). Exposures of mice to 1000 ppm continuously for 14 weeks decreased activity levels (Thomas et al. 1972 as cited in USEPA 1985).

CHRONIC TOXICITY

Chronic methylene chloride exposure has been associated with hepatotoxic and nephrotoxic effects. Inhalation exposure to 100 ppm methylene chloride for 100 days resulted in slight cytoplasmic vacuolization in the liver with

positive fat stains, and tubular degeneration in the kidneys of rats and mice (Hann et al. 1972 as cited in USEPA 1985). Similar changes in the liver were found in dogs and monkeys exposed for 4 weeks to 1000 ppm methylene chloride. Dogs, cats and guinea pigs have exhibited liver injury following exposures in the range of 7 200 to 10 000 ppm for periods of from 4 to 75 weeks (Heppel et al. 1944 as cited in USEPA 1985; Lehman and Flury 1943 as cited in ACGIH 1981). Repeated methylene chloride exposure to 500 ppm to 5000 ppm has also been shown to increase the metabolic activity of rat liver microsomes (Norpoth et al. 1974 as cited in USEPA 1985).

Exposure of rats and mice to 1250 ppm methylene chloride 7 h/d during days 6-15 of gestation resulted in an increased incidence of dilated renal pelvis among rat pups and an increased incidence of extra sternebrae among mice pups (Schwetz et al. 1975 as cited in USEPA 1985). Exposure of pregnant rats to 4500 ppm methylene chloride 6 h/d, 7 d/wk during pregnancy resulted in decreased body weights among the fetuses (Hardin and Manson 1980 as cited in USEPA 1985). These offspring also showed retarded behavioral habituation to novel environments (Bornschein et al. 1980 as cited in USEPA 1985).

Methylene chloride is considered to be an animal carcinogen. A dose-related increase in incidence of salivary gland region sarcomas was observed in male rats following 2 years of inhalation exposure to 1500 ppm or 3500 ppm methylene chloride for 6 h/d 5 days per week (Dow Chemical Co. 1980 as cited in USEPA 1985). Male mice receiving about 235 mg/kg/d methylene chloride in the drinking water for 24 months had a very slightly increased incidence of hepatocellular adenomas and/or carcinomas (National Coffee Assoc. 1983 as cited in USEPA 1985). In a National Toxicology Program sponsored

investigation, 500 or 1000 mg/kg/d methylene chloride was administered by gavage to both male and female Fischer 344 rats and $B6C3F_1$ mice for 2 years. Increased incidences of hepatic neoplastic nodules and adrenal cortical adenomas were observed in both rat sexes, and an increased incidence of pancreatic acinar-cell adenomas was found in males. Hepatocellular carcinomas were observed in both sexes of treated mice (NTP 1982 as cited in NRC 1983). These results however are undergoing re-evaluation as the National Toxicology Program Technical Review Subcommittee concluded that the maximum tolerated dose may have been exceeded in these studies (NRC 1983). An additional National Toxicology Program sponsored bioassay exposing Fischer 344/N rats and B6C3F1 mice to 0, 1000, 2000 or 4000 ppm is currently underway (NTP 1984b).

UCR

EPA's Carcinogen Assessment Group has estimated a unit risk of 6.3×10^{-4} $(mg/kg/d)^{-1}$ methylene chloride, based on carcinogenicity data from oral animal studies (CAG 1984).

ACUTE TOXICITY

The acute toxic effects of methylene chloride on humans include decreased psychomotor performance (Winneke 1974, as cited in USEPA 1980), central nervous system dysfunction and irritation of the mucous membranes (eyes, respiratory tract, and skin) (NAS 1978, as cited in USEPA 1980). Mild poisoning produces somnolence, lassitude, anorexia, and mild lightheadedness. Fatal poisonings have resulted from cardiac injury and heart failure (NAS 1978, as cited in USEPA 1980). Upon metabolism, methylene chloride will form carbon monoxide, which will increase carboxyhemoglobin levels in the blood.

Several investigators have noted the potential risks of elevated carboxyhemoglobin levels to individuals with pre-existing cardiovascular stress (e.g., angina and myocardial ischemia) (NAS, 1980).

Virtually all experimental acute exposure studies of methylene chloride using humans have studied neural or behavioral dependent variables (USEPA, 1983). Stewart et al. (1972b as cited in USEPA, 1983) reported that subjects had symptoms of "lightheadedness" and had difficulty with speech articulation at methylene chloride levels higher than 3012 mg/m^3 for more than one hour. The investigators also reported anecdotal data about increased amplitude in certain visual evoked potentials at about the same exposure levels; however, no statistical analyses were possible because of the small number of subjects (three to eight individuals). In a later study (Stewart el al., 1973, as cited in USEPA, 1983) they reported that 173, 347 or 867 mg/m^3 methylene chloride had no effect on the Romberg equilibrium test, vigilance, coordination or arithmetic skills.

Winneke (1974) and Winneke and Fodor (1976) (both as cited in USEPA, 1983) reported that the central nervous system was impaired during methylene chloride exposure. These investigators exposed female human subjects to 0, 1041, 1730 and 2776 mg/m^3 methylene chloride for up to three hours. They measured performance on a series of hand-eye coordination tasks. They also measured the critical flicker fusion (CFF) frequency and performance on an auditory signal detection task. Concentration related decrements in signal detection, beginning at 1041 mg/m^3 were observed. Many motor decrements were observed at 2776 mg/m^3, the only concentration at which these tests were conducted. Fodor and Winneke (1971, as cited in USEPA, 1983) reported a

similar study in which exposure to 1044 and 2776 mg/m^3 resulted in both decreased CFF frequency and impaired signal detection behavior; however, the two methylene chloride levels did not produce effects significantly different from each other.

Putz et al. (1976, as cited in USEPA, 1983) exposed six men and six women (ages 18 to 40 years) to 700 mg/m^3 of methylene chloride for four hours. Eye and hand coordination at the end of the exposure period were depressed as compared with controls. Peak alveolar carbon monoxide after the four hours was 60 mg/m^3 and carboxyhemoglobin was 5.1 percent. These levels are similar to the levels observed for four hour exposures to carbon monoxide, causing the authors to conclude that carbon monoxide may have been primarily responsible for the decreases in performance after exposure. (Other experiments have shown that methylene chloride is more toxic than carbon monoxide (NAS, 1980; USEPA, 1983)).

Gamberale et al. (1975, as cited in USEPA, 1983) studied the effect of methylene chloride exposure on psychomotor and cognitive performance in 14 men, ages 20 to 30 years. The men were exposed for successive 30 minute intervals of 870, 1740, 2600 and 3470 mg/m^3 (half the men were exposed to increasing levels, half to decreasing levels). Total exposure time was two hours. During the exposure, no statistically significant impairment in any measured performance was observed, although reaction time appeared to be affected.

Stewart et al. (1972a, b, as cited in USEPA, 1983) reported no effects of exposure to 1730 or 3460 mg/m^3 methylene chloride for two hours on various hematological, hepatic or renal measures. Continuous electrocardiographic

monitoring during one to two exposures (duration unspecified) to 3460 mg/m^3 did not reveal arrhythmias. Gamberale et al. (1975, as cited in USEPA, 1983) reported that methylene chloride levels up to 3460 mg/m^3 produced no alterations in heart rate (duration of exposure unspecified).

Stewart and Dodd (1964, as cited in USEPA, 1983) studied groups of three to five men and women (25 to 62 years old) who immersed their thumbs in various chlorinated aliphatic solvents. Within the first 2 minutes of immersion in methylene chloride, all subjects reported an intense burning sensation on the dorsal surface of their thumbs. Within 10 minutes, a feeling of coldness or numbness, alternating with the burning sensation, was reported; the slightest movements of the thumb triggered waves of searing and excruciating pain. Following removal from the solvent, their thumbs were described as "numb and cold" or "asleep." Within one hour, both the erythema and paresthesia subsided.

Studies of human blood <u>in vitro</u> from individuals homozygous for sickle cell anemia (Matthews et al., 1977, 1978, as cited in USEPA, 1983) have shown that methylene chloride caused hemolysis, preferentially of sickle and other abnormal cell types. These reports suggest that acute exposure to relatively high concentrations of methylene chloride may result in anemia, particularly in individuals who are predisposed to hemolytic anemias.

In addition to the experimental studies discussed above, human toxicity data are available from reports of accidental exposure to methylene chloride. The lowest recorded lethal dose for humans is 500 mg/kg (NIOSH, 1978, as cited in USEPA, 1981). However, quantitative data on exposure levels is usually not

available in these cases. Human case studies involving myocardial infarction have been reported from methylene chloride exposure and have included fatalities. For example, methylene chloride was a probable factor in the death of a worker who became unconscious, fell in a vat containing methylene chloride, and died after suffering myocardial infarctions (Kuzelova et al., 1975, as cited in USEPA, 1983). However, Torkelson and Rowe (1981) report that experience with use of methylene chloride has generally been favorable. According to these authors, six reports of death have been found and these deaths were the result of gross over exposure. They state that, most commonly, recovery even following exposure to anesthetic concentrations, has been rapid. Non-fatal exposures have caused electrocardiogram changes, similar to those induced by carbon monoxide (USEPA, 1983). A variety of neurotoxic effects have also been reported (NIOSH, 1976). USEPA (1983) states that the case histories of certain affected exposed individuals suggest that the individuals had pre-existing cardiovascular disease.

Hepatotoxicity has not been reported in any human case report, even following fatal exposures. The only evidence of human nephrotoxicity was a finding of congested kidneys following a fatal exposure. Ocular toxicity other than eye irritation and congested conjunctivae has not been reported (USEPA, 1983).

ASEL

Based on the data presented in the toxicity profile, the acute exposure level (ASEL) i.e., the dose below which adverse health effects would not be expected to occur) for acute exposures, was calculated to be 2.5 mg/kg.

This level of 2.45 mg/kg was based on inhalation studies in humans in which very minor auditory effects were reported. Specifically, one study noted dose-related decrements in auditory signal detection tests when women are exposed to 1041 mg/m^3 methylene chloride for three hours. Assuming that an average woman weighs 60 kg and breathes 16 L/min (at light work) the dose to the women in this study was calculated as follows:

$$\text{Dose} = 1041 \frac{mg}{m^3} \times \frac{L\ m^3}{1000\ L} \times \frac{16\ L}{min} \times \frac{1}{60\ kg} \times 180\ min = \underline{50\ mg/kg}$$

A safety factor of 10 is appropriate in this case because this level produced minor effects and a factor of 10 should account for the fact that humans vary in susceptability to the minor effects of chemicals. An additional safety factor is also used when estimating a no effect level from a lowest observed effect level. In this case, a safety factor of 2 is appropriate because of the relatively minor effects observed. The acute safe exposure limit based on this study would therefore be 2.5 mg/kg.

STPL

In the case of methylene chloride, the NAS has not established short-term exposure limits. We will therefore apply the criteria established by NAS (1979) to calculate a STPL for the rare occurrence of a methylene chloride spill. Because of the large amount of human data available it can clearly be established that short-term exposure to methylene chloride, at levels of 1000 ppm or less, are not associated with any irreversible effects on the heart, liver, kidney, or blood (Stewart et al 1972a, b). All other reported effects from short-term exposures are nonincapacitating reversible neurological

effects (Table A-7). None of these neurological effects would be expected to be so severe that they would impair the ability of individuals to leave an area if needed. Short-term exposure to levels of 868 ppm was only found to cause lightheadedness while levels of 1000 ppm were found to have no effect on psychomotor or cognitive measures. The lowest level found to affect eye-hand coordination was 200 ppm (Puty et al 1976), but 250 ppm was not found to affect vigilance, equilibrium coordination, or arithmetic skills (Stewart et al 1973).

Given the lack of any organ toxicity at 1000 ppm, coupled with the lack of serious psychomotor and cognitive impairment, ENVIRON recommends that an STPL of 200 ppm (706 mg/m^3) for no more than 1 hour be used. This is based on the use of a small (5X) uncertainty factor because of the numerous human studies that have been conducted with methylene chloride.

TABLE A-7

HUMAN EFFECTS REPORTED AFTER
SHORT-TERM EXPOSURE TO VARIOUS LEVELS OF METHYLENE CHLORIDE

Finding	Dose, mg/m^3 (ppm)	Citation*
No effect on psychomotor and cognitive measure	3470 (999)	Gamberale et. al., 1975
No effect on hematology hepatic, or renal measures	3460 (996)	Stewart et. al., 1972a,b
"Lightheaded"	3017 (868)	Stewart et. al., 1972b.
Effects on Hand eye coordination	2776 (799)	Winneke and Fodor, 1976
Effects on signal diction and critical flicker fusion	1041 (300)	Winneke; 1971 Winneke and Fodor, 1976
No effect on equilibrium, vigilance, coordination, arithmetic skills	867 (250)	Stewart et. al. 1973
Effect on eye hand coordination	700 (200)	Puty et. al. 1976.

*All as cited in EPA 1983

PETROLEUM DISTILLATES

Petroleum distillates, as defined here, consist of naphtha (rubber solvent), mineral spirits (petroleum spirits, Varsol), and Stoddard solvent. Each component of petroleum distillates is itself a complex mixture of hydrocarbons with toxicity dependent upon the attendant toxicities of the constituents in the mixture. For each component, there is also the potential for synergistic effects. The predominant adverse effects from exposure to petroleum distillates are eye, nose, and throat irritation, dermatitis, and central nervous system depression.

NAPHTHA (rubber solvent)

Naphtha has a boiling range of 45-125°C and consists primarily of hydrocarbons containing five to seven carbons (C_5 - C_7). Fifteen-minute exposures to human volunteers caused slight transitory irritation at a vapor concentration of 430 ppm and eye irritation and headache at 2000 ppm (NIOSH, 1977). In animals, a vapor concentration of 24 200 ppm produced convulsions followed by death after a 4 hour exposure; impairment of motor coordination and eye irritation was observed at 5300 ppm and 9800 ppm, respectively (NIOSH, 1977). In dogs and rats exposed to naphtha up to 480 ppm for 6 hours per day, 5 days per week for 63 days, no adverse effects were detected (ACGIH, 1980). Using the animal NOEL from this study and applying a safety factor of 1000, a toxicity reference dose of 0.20 mg/kg/d is recommended for the ingestion route of administration.

MINERAL SPIRITS (petroleum spirits, Varsol)

Mineral spirits has a boiling range of 150 to 200°C and consists approximately of 85% aliphatic hydrocarbons and 15% aromatic hydrocarbons. Fifteen men

exposed to mineral spirits at a concentration of 1440 or 2880 ppm for an unspecified duration experienced nausea and vertigo (dizziness) (NIOSH, 1977). Human subjects exposed to 360, 720, 1080, and 1440 ppm in 4 consecutive 30 minute periods resulted in no impairment in tests for perceptual speed, numerical ability, manual dexterity, reaction time, or short-term memory (NIOSH, 1977). Eight human subjects exposed to mineral spirits at a concentration of 2300 ppm for 50 minutes resulted in a prolongation in reaction time, a probable impairment in short-term memory, but no impairment in the other three tests. Rector et al. (1966, cited in NIOSH, 1977) exposed rats, guinea pigs, rabbits, dogs, and monkeys to mineral spirits continuously for 90 days at concentrations from 65 to 730 ppm. A decline in body weight was detected in guinea pigs and monkeys at 350 and 320 ppm, respectively. A significant number of deaths occurred only in guinea pigs. Upon microscopic examination of surviving animals, no evidence of toxicity was apparent in heart, spleen, kidney, or liver. However, lung congestion, bronchitis, and mixed inflammatory cell infiltration was observed in all species exposed to the highest concentration of mineral spirits tested (730 ppm). Using the monkey LOEL of 320 ppm, a safety factor of 1000, and a severity factor of 2.14 a toxicity reference dose of 0.10 mg/kg/d is recommended for the ingestion route of administration.

STODDARD SOLVENT

Stoddard solvent is generally considered to be one type of mineral spirits. It has a boiling range of 160 to 210°C and is composed primarily of hydrocarbons containing seven to twelve carbons (C_7 - C_{12}). Six human volunteers exposed to Stoddard solvent for 15 minutes experienced no irritation at 24 ppm, slight transitory eye irritation in one of the subjects

at 150 ppm and in all six subjects at 470 ppm (NIOSH, 1977). Cases of aplastic anemia as a result of intermittent long-term exposure to Stoddard solvent has been reported (NIOSH, 1977). Fifteen rats exposed to 1400 ppm for 8 hours resulted in eye irritation, slight loss of coordination, a bloody exudate around the nostrils, and one death (NIOSH, 1977). At 800 ppm, similar effects resulted except there was no loss of coordination. At 400 ppm, no adverse effects were detected. In rats and dogs exposed to 0, 84, 190, and 330 ppm for 6 h/d, 5 days per week for 13 weeks, various hematological changes were observed in rats but the changes were not dose-dependent (NIOSH, 1977). Kidney damage was observed in rats exposed to the highest concentration (330 ppm). In another study, rats, guinea pigs, rabbits, dogs, and monkeys exposed continuously to 290 ppm for 90 days resulted only in minor lung congestion and emphysema in guinea pigs (NIOSH, 1977). Based on the animal LOEL of 290 ppm in guinea pigs and applying a safety factor of 1000 and a severity factory of 2.14, a toxicity reference dose of 0.41 mg/kg/d is recommended for the ingestion route of administration.

The toxicity reference doses for each of the components of petroleum distillates are very similar. They are 0.20 mg/kg/d for naphtha, 0.10 mg/kg/d for mineral spirits, and 0.41 mg/kg/d for Stoddard solvent. The lowest of the three, 0.10 mg/kg/d, is selected to represent the toxicity reference dose for petroleum distillates.

ASEL

Humans exposed acutely to 400 ppm of Stoddard solvent showed no adverse effects (Patty, 1981). In one study, humans exposed to 350 ppm of mineral spirits for 8 hours suffered mucous membrane irritation, and in another study,

30 min exposure to 670-1670 ppm caused nausea, vertigo (loss of balance) and some cardiac irregularities (Patty, 1981). Using 670 ppm as the acute LOEL and applying a safety factor of 20 to protect the sensitive segments of the general population against these effects, the recommended acute safe exposure level (ASEL) is 34 ppm.

STPL

Using the same studies described above for setting the ASEL, a STPL of 67 ppm is proposed based on the acute LOEL of 670 ppm and applying a ten-fold safety factor. This should provide an adequate margin of safety to prevent the occurrance of irreversible adverse effects after short-term exposure to petroleum distillates.

PHENOL

Phenol is readily absorbed by humans by all routes of exposure. Following absorption, the compound is rapidly distributed to all tissues. The predominant effects in humans following chronic exposure are digestive disturbances, effects on the central nervous system, and, possibly skin disorders (Deichmann and Keplinger 1981, USEPA 1980).

Prolonged oral exposure in animals can cause damage to the lung, liver, kidneys, heart and genitourinary tract. Chronic inhalation induces respiratory difficulties, lung damage, loss of weight and paralysis (Deichmann and Keplinger 1981). Rats exposed to phenol in their drinking water for 6 months at various levels exhibited no significant effects at concentrations of 5000 ppm or below. At 7000 ppm, depressed growth occurred in the young (Heller and Purcell 1938). Reduced weight gain developed in rats drinking phenol in water for 12 months (USEPA 1980f). Phenol is not a carcinogen when administered alone; however, when applied to the skin of mice after initiation with known carcinogens, phenol appears to promote the development of tumors. Mutagenicity and teratogenicity have not been thoroughly examined (USEPA 1980f).

Rats were administered 50 or 100 mg/kg/d phenol by gavage over a 6-month period (135 doses). After this period, effects on liver and kidney tissue appeared. Slight liver changes and slight to moderate kidney damage were found in rats receiving 100 mg/kg; slight kidney damage occurred in the 50 mg/kg group (Dow Chemical Co. 1976 as reported in USEPA 1980f). The USEPA has derived an ADI for phenol of 0.1 mg/kg/d based on kidney damage in rats (USEPA 1984).

POLYCHLORINATED BIPHENYLS

Polychlorinated biphenyls (PCBs) produce severe toxic effects in man and animals, particularly following repeated exposure by inhalation and ingestion. Major target organs are the skin and liver; however, the gastrointestinal tract and the nervous system are also affected (USEPA 1980e, Deichmann 1981). In human males occupationally exposed to Aroclor 1254, a slight increase in the incidence of cancer, particularly melanoma of the skin, has been reported (Deichmann 1981). This finding however, has not been confirmed.

Female mink fed Aroclor 1254 for 9 months failed to produce offspring (NRC 1977). Rats receiving oral doses of Aroclor 1254 for 18 months showed an increase in liver weight and a reduction in litter survival (NRC 1977). Male Sprague-Dawley rats fed Aroclor 1254 for 52 weeks showed biochemical changes accompanied by morphologic alterations in the liver (NRC 1980). Reproductive effects were noted in rats and chickens when fed Aroclor 1254 (Deichmann 1981). In rabbits, Aroclor 1254 produced teratogenic effects when ingested (Deichmann 1981). The offspring of rhesus monkeys fed PCBs (Aroclor 1248) at 0.5 ppm in the diet on 3 days per week throughout pregnancy and during nursing were underweight at birth, and were hyperactive and showed impaired learning ability when tested at 6 months of age (Bowman et al. 1981). This dietary concentration corresponds to about 0.01 mg/kg/d.

Several rodent studies suggest that some PCBs are carcinogens and may enhance the carcinogenicity of other chemicals (USEPA 1980e). Aroclor 1260

induced a significant increase in the incidence of hepatocellular carcinoma and neoplastic nodules in female rats fed 100 ppm in the diet for 21 months (Kimbrough et al. 1975).

EPA's Carcinogen Assessment Group estimates a unit risk for polychlorinated biphenyls of 4.3 $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984). This value will be used for our risk assessment. The Ontario Ministry of the Environment (OMOE, 1984) also published a cancer risk assessment on PCB's but we will not use this estimate of the UCR for this assessment because the MOE risk assessment leads to safe exposure levels which are at least 50 times larger than obtained using the CAG (1984) UCR. Several factors contribute to the differences in risk estimates between MOE and CAG, which are outlined below:

Source of Difference	MOE Procedure	CAG Procedure
Data set	Hepatocellular carcinoma only from Kimbrough study	Hepatocellular carcinoma or neoplastic nodule from Kimbrough study
Extrapolation model	Linear extrapolation from 99% upper confidence limit on carcinoma incidence	Application of linearized multistage model to carcinoma/neoplastic nodule incidence
Interspecies extrapolation assumption	Rats and humans assumed at equal risk at same dietary concentration of PCB	Rats and humans assumed at equal risk at same daily dose level in mg/m^2 body surface area/day.

These three differences together account for over a 50-fold difference in risk estimates.

PYRIDINE

Human exposure to pyridine is usually by inhalation. Effects of exposure include transient effects on the central nervous system (CNS) and the gastrointestinal tract. Common symptoms of exposure are headache, dizziness or giddiness, nervousness, insomnia, mental dullness, nausea and anorexia. Pyridine is also a photosensitizer (Clayton and Clayton 1981). The oral LD_{50} in rats is 891 mg/kg (NIOSH 1983).

In animal studies, pyridine is not carcinogenic or mutagenic. In testing for teratogenicity, pyridine injected into chicken eggs causes typical muscular hypoplasia of the legs (Clayton and Clayton 1981).

Exposure of rats for 7 hours per day, 5 days per week for 6 months to either 10 or 50 ppm pyridine vapor did not change either the growth rate or mortality when compared with controls. However, increased liver weight to body weight ratios were noted in the treated rats (EOHS 1971). At vapor concentrations of 6 to 12 ppm in occupational exposure studies, mild symptoms of CNS injury have been noted (Teisinger 1948).

Exposure of rats for 7 hours per day, 5 days per week for 6 months to either 10 or 50 ppm pyridine vapor did not change either the growth rate or mortality when compared with controls. However, increased liver weight/body weight ratios were noted in the treated rats (EOHS 1971). An ADI of 9.0×10^{-3} mg/kg/d was estimated using this study by applying safety factors of 100 for inter and intraspecies variation and 5 for the increased liver weight to body weight ratios noted.

STYRENE

The major effect of acute exposure to styrene is eye, skin respiratory system and mucous membrane irritation. Acute exposure of humans to styrene results in similar irritation symptoms in addition to symptoms such as nausea, vomiting and neurological and psychological disturbances (Stewart et al. 1968 and Rogers and Hooper 1956, as cited din Sandmeyer 1981). Orally, styrene has low toxicity; the oral LD_{50} for rats is 5 g/kg. By inhalation, the approximate LC_{50} is 6000 ppm for a 4 h exposure in rats.

A no effect level for chronic exposure to styrene of between 66.7 and 133 mg/kg/d has been reported (Wolf et al. 1956 and Spencer et al 1942, as cited in Sandmeyer 1982). The higher dose resulted in decreased growth and some liver and kidney organ weight deviation. At 1000 mg/kg/day, mortality was increased.

Occupational studies conducted in Russia indicate that additional symptoms, such as anemia, leukopenia, pancreatic changes, and gastric acidity reduction may also be associated with styrene exposure (Bashisov, 1970 and Volkova et al 1970 as cited in Sandmeyer 1981). Some CNS defects have been observed in children whose mother had been exposed to chemicals such as styrene during pregnancy (Sandmeyer 1981). All of these symptoms however are nonspecific and may relate to solvent effects (Sandmeyer 1981).

Styrene has been shown to increase the incidence of lung tumors in offspring of mice given styrene during pregnancy (Ponomarkov and Tomatis 1979, as cited in IARC 1979). Chromosomal aberrations have been noted in workers

exposed occupationally to styrene (Meretoja et al. 1977 as cited in IARC 1979). However, it has yet to be shown that styrene is carcinogenic in humans.

The toxicity reference was estimated using the no effect level of 133 mg/kg/d reported by Wolf et al. (1956 as cited in Sandmeyer 1981). A safety factor of 100 to account for intra and interspecies variation was applied to the daily dose of 95 mg/kg/d, resulting in a ADI of 9.5×10^{-1} mg/kg/d.

TETRACHLOROETHYLENE

Several recent literature reviews have been published regarding the toxicity of tetrachloroethylene (NRC 1977, 1980, 1983; USEPA 1980; Torkelson and Rowe 1981 ACGIH 1980 and IARC 1979). The following information has been summarized from these reviews. Acute inhalation exposures among humans are characterized by central nervous system depression and possibly delayed kidney and liver dysfunction and/or pathology. Symptoms of acute exposure to about 100 ppm for two hours include light headedness, burning in the eyes, congestion, and difficulty in motor coordination. Dizziness, headache, unconsciousness and death can occur as the acute exposure level approaches 2000 ppm. Acute exposures of humans are also sometimes followed by elevations in clinical chemistry measures of liver dysfunction and mild hepatitis.

Central nervous system depression follows acute exposure of laboratory animals to tetrachloroethylene as well, along with abnormal weakness, handling intolerance, restlessness, irregular respiration, muscle incoordination and unconciousness. The minimum narcotic concentration in mice is 2950 ppm and the minimum lethal dose is 6000 ppm. Slight increases in liver weights, total lipid and liver enzymes follow short exposures of animals to concentrations in the 1000 to 6000 ppm ranges, and a dose-related fatty infiltration of the liver has been reported following a 4 hour exposure of mice to 200 to 1600 ppm tetrachloroethylene. Oral LD_{50}'s for rats and mice are 2600 to 1300 mg/kg and 8850 mg/kg respectively. A 4 hour LC_{50} for mice of 5200 ppm and an 8 hour LC_{50} for rats of 5040 ppm have been reported.

Prolonged exposure of humans to tetrachloroethylene leads to irritation of the respiratory tract, nausea, headache, sleeplessness, abdominal pains and constipation. Sustained ambient atmospheric concentrations of greater than 200 ppm can lead to narcosis and death. Workers exposed to between 100-400 ppm tetrachloroethylene in dry cleaning plants exhibit central nervous system depression, dysfunction of the autonomic nervous system, slowed motor nerve conduction velocity and alterations in the EEG. Hepatotoxic effects, which include enlarged liver, cirrhosis of the liver, hepatitis, and increases in serum levels of liver enzymes, and nephrotoxic effects which include uremia, proteinuria and increases in serum creatinine, have also occassionally been reported among chronically exposed populations.

Liver effects predominate in animal studies of chronic tetrachloroethylene exposure. Rats exposed to 600 ppm 6 h/d, 5 days per week for 12 months showed reversible toxic effects in the liver; rats exposed to 230 and 470 ppm 8 h/d, 5 days per week for up to 7 months showed pathological effects in both kidney and liver; and, rats exposed to 1600 ppm 7 h/d for 183 days exhibited enlargement of both kidney and liver. Mice exposed to 200 ppm 4 h/d, 6 days per week for 1, 2, 4 or 8 weeks showed fatty degeneration in the liver, but no liver necrosis and no effects on the kidney. Guinea pigs appear to be especially sensitive exhibiting increased kidney weight and increased liver weight with fatty degeneration after 400 ppm 7 h/d for 183 days, and increases in liver weight after 100 ppm for the same amount of time. Mice also show increased liver weights and hepatocellular swelling in the centrilobular region after 11 days of 100 or 250 mg/kg administered orally.

Excess toxic nephropathy is observed in rats exposed to about 500 or 1000 mg/kg by oral gavage 5 days per week for 78 weeks.

Mice exposed to 300 ppm tetrachloroethylene 7 h/d on days 6 through 15 of gestation exhibit decreased fetal body weight, an increased incidence of subcutaneous edema and increased incidence of delayed ossification of skull bones and split sternebrae. Pregnant rats exposed according to the same paradigm showed an increased incidence of resorptions. There was a reduction in the number of pups born alive among rats exposed to 100 ppm tetrachloroethylene 7 h/d on days 7 through 13 or on days 14 through 20 of gestation. Surviving offspring showed initial neurobehavioral deficits which disappeared with age and some changes in neurotransmitter level which persisted at least until 21 days of age.

Tetrachloroethylene (25-100 μmol/egg) injected into the air space of 5 day old developing chick embryos caused a dose related increase in mortality, and those chicks that survived exhibited excess skeletal anomalies.

Tetrachloroethylene is believed to have a low potential for carcinogenicity in mice. B6C3F1 mice exposed by oral gavage to a time weighted average of 536 or 1072 mg/kg (males) and 386 or 772 mg/kg (females) 5 days per week for 78 weeks showed an increased incidence of hepatocellular carcinomas. Rats exposed to 471 or 941 mg/kg (males) and 474 or 949 mg/kg (females) according to the same paradigm showed no carcinomas. However, these rats suffered from a high rate of mortality due to the early development of toxic nephropathy among experimental subjects, rendering interpretation of the negative response inconclusive. Moreover, the positive control, carbon tetrachloride treated rats, showed a poor response in this study.

EPA's Carcinogenic Assessment Group has estimated a unit risk for tetrachloroethylene of 3.5×10^{-2} $(mg/kg/d)^{-1}$, based on data from animal oral studies (CAG 1984).

TETRAETHYL LEAD

Exposure of experimental animals to high concentrations of tetraethyl lead (TEL) has resulted in CNS effects such as irritability, hyperactivity, tremors, spasticity, arched backs and postration (Stokinger 1981). The oral LD_{50} in rats is 17 mg/kg, and the one-hour LC_{50} is 0.85 mg/kg (ACGIH 1983).

In chronic exposures in rats, TEL accumulates in the blood, liver and kidney (Stokinger 1981). Histologic changes occur in the liver, pancreas, anal and endocrine systems by oral, inhalation or cutaneous routes of administration (ACGIH 1983). Schepers (1964 as cited in ACGIH 1983) fed rats tetraethyl lead at concentrations of 0.17 mg and 0.0017 mg/kg five times per week for 21 weeks. Histologic changes, but no changes in body weight were observed.

In humans, TEL intoxication is characterized by insomnia, excessive dreaming, emotional instability and increased erratic physical activity (IARC 1973). No studies to assess carcinogenicity of TEL have been reported.

The data reported by Schepers (1964 as cited in ACGIH 1981) were used to estimate a toxicity reference dose. Safety factors of 100 for intra and interspecies variability, 10 for extrapolation from subchronic exposure, and 5 for the histologic changes observed were applied to the average daily dose of 0.0012 mg/kg/d, resulting in an ADI of 2.4×10^{-7}.

TOLUENE

Toluene is readily absorbed by inhalation and ingestion but less well absorbed through the skin. In humans, inhalation exposure can cause central nervous system (CNS) depression. Symptoms of exposure include altered psychomotor performance, irritability, disorientation and unconsciousness (NAS 1980). Some effects of narcosis are evident at around 200 ppm (NAS 1977). Workers exposed to levels of less than 200 ppm complain of headaches, lassitude and nausea; however, no physical effects occur (ACGIH 1981). Women occupationally exposed to toluene report a high incidence of menstrual disorders as well as effects on embryonic and fetal development (NAS 1980).

In laboratory animals, a 4 h LD_{50} of 8000 to 8800 ppm and a minimum 8 hour lethal concentration of 5300 have been reported.

In animals, subtle cytological and clinical changes as well as behavioral changes occur following chronic inhalation exposure (Clayton and Clayton 1981). Dogs exposed 8 h/d, 6 days per week for 6 months to 2000 to 2660 ppm toluene exhibit intoxication, incoordination and hind leg paralysis. Other effects such as thrombocytopenia, leukocytosis, and chromosome damage in bone marrow cells occur in mice and rats following inhalation of low levels of toluene; however, the purity of the toluene used in those latter studies is questionable (USEPA 1980). When applied to the skin of rats, toluene produces hematological changes (NAS 1980). Toluene is not mutagenic or carcinogenic. Teratogenicity occurs in mice (USEPA 1980).

The USEPA has derived an ADI for 4.9×10^{-1} mg/kg/d for toluene based on central nervous system effects (USEPA 1984).

1,1,1-TRICHLOROETHANE

Several recent literature reviews have been published regarding the toxicity of 1,1,1-trichloroethane (IARC 1979; NRC 1980, 1983; USEPA 1980, 1981, ACGIH 1980 and Torkelson and Rowe, 1981). The following information has been summarized from these reviews.

The primary effect of acute 1,1,1-trichloroethane exposure is depression of the central nervous system. Psychophysiological functions are hindered at concentrations of 350 ppm and eye irritation and dizziness occur at 450 ppm. Anesthetic effects occur at concentrations approaching 500 ppm and death is likely to occur at concentrations in excess of 14 000 to 15 000 ppm. Fatalities which have occurred have been attributed to suffocation and/or acute cardiac arrhythmia. Pulmonary congestion and edema, and mild hepatoxic effects which include fatty vacuolation and elevated urinary urobilinogen, have also been associated with acute 1,1,1-trichloroethane inhalation.

Similar anesthetic and cardiac responses have been reported in animals following acute exposures to 1,1,1-trichloroethane. Central nervous system depression rapidly follows exposure of rats to 18000 ppm, which is the 3 hour LC_{50}. Cardiac arrhythmias follow exposure of dogs to 1,1,1-trichloroethane in the range from 5000 to 10 000 ppm. The oral LD_{50} in mice and rats is about 11 000 mg/kg body weight, the i.p. LD_{50} is 5000 mg/kg. Liver damage has been reported in rats only after exposure to nearly lethal doses.

Animals appear to be able to survive repeated exposures to very high levels of 1,1,1-trichloroethane. Rats exposed to 10000 ppm 1,1,1-trichloroethane 7 h/d 5 days per week for 1 to 3 months, showed

staggering gait and weakness after 10 minutes and by 3 hours were semiconscious but survivors had completely recovered the following morning. All rats exposed to 5000 ppm 7 h/d 5 days per week survived over 31 exposures.

Repeated inhalation exposures below 1000 ppm appear to have little discernible toxicological effect. Mice exposed continously to 1000 ppm for 14 weeks showed signficant histopathological effects in the liver as well as increased liver weight and triglyceride content. Fatty changes increased liver weight were also observed among guinea pigs exposed to 1000 ppm daily for 90 days. Exposure of rabbits and dogs to 370 ppm daily for 90 days depressed their body weight. Exposure of rats to 800 ppm for 4 weeks increased liver weights but had little other toxic effect.

Rats administered 750 or 1500 mg/kg by gavage and mice administered time weighted averages of 2800 or 5600 mg/kg 5 days per week for 78 weeks leads to diminished body weight gain and decreased survival time, but no specific histopathological changes which can be identified.

1,1,1-trichloroethane does not appear to interfere with reproductive processes or to have teratogenic effects. No effects on average litter size, average number of implantation sites per litter, incidence of fetal resorptions or fetal body measurements were observed among rats or mice exposed to 875 ppm 1,1,1-trichloroethane vapor 7 h/d from days 6 through 15 of gestation. Similarly, no effects on conception and no toxic effects were observed among offspring of rats exposed to 21000 $\pm$ 200 ppm 1,1,1-trichloroethane from two weeks before breeding until day 20 of gestation, or among mice exposed to 1000 mg/kg in their drinking water.

A preliminary study by NTP (1983) indicates a compound-related increased incidence of hepatocellular carcinomas. Male and female mice were administered the compound by gavage at doses of 0, 1500 and 3000 mg/kg/d. The combined incidences of hepatocellular adenomas or carcinomas in male mice was 5/49, 7/49 and 12/499 in control, low dose and high dose groups, respectively. The Carcinogen Assessment Group of EPA has estimated a unit risk value for 1,1,1-trichloroethane of 1.6×10^{-3} $(mg/kg/d)^{-1}$ (CAG 1984).

TRICHLOROETHYLENE

Several recent literature reviews have been published regarding the toxicity of trichloroethylene (NRC 1977, 1980, 1983; Torkelson and Rowe 1981, USEPA 1980, ACGIH 1980 and IARC 1979). The following information has been summarized from these reviews.

In humans, acute exposure to trichloroethylene leads primarily to central nervous system depression. Other reported symptoms include visual disturbances, mental confusion, fatigue, tremors, dizziness, nausea and vomiting. High doses also lead to acute renal failure, anuria, uremia and hepatic and cardiovascular damage. Death is frequently attributed to ventricular fibrillation and cardiac arrest. The lowest reported oral lethal dose has been 50 mg/kg. The lowest reported inhaled lethal dose has been 3000 ppm for 10 minutes although concentrations higher than this are often nonfatal.

A low acute toxicity of trichloroethylene is apparent among laboratory animals with the major effect being mild hepatotoxicity. The oral LD_{50} in rats is 4920 mg/kg; in mice is 3200 mg/kg and in dogs is 2800 mg/kg. At near lethal oral doses, mild hepatic dysfunction has been reported in rats. Central nervous system symptoms predominate following high short inhalation exposures although hepatic injury has been reported in rats following a 2 hour exposure to 10000 ppm trichloroethylene. Rats exposed for several hours to 200, 400 to 800, or 1600 ppm trichloroethylene exhibited an increase in spontaneous motor activity, inhibited swimming, and decreased motor activity respectively.

Chronic exposure of laboratory animals to trichloroethylene vapor appears to result in mild hepatotoxic and nephrotoxic effects. Increased liver weight has been observed in rats, guinea pigs and rabbits exposed to 400 ppm trichloroethylene 7 h/d, 5 days per week for 6 months. Increased kidney weights were observed in rats and rabbits if the exposure level was 3000 ppm. Rats exposed to 55 ppm trichloroethylene 8 h/d, 5 days per week for 14 weeks also have increased liver weights, and mice exposed to 1600 ppm 7 to 8 h/d 6 days per week for 8 weeks show a slight accumulation of lipids in the liver.

Histological changes in the brain, especially the cerebellum, have been reported in dogs following exposure to 500 to 3000 ppm trichloroethylene 2 to 8 h/d 5 days per week for a total of up to 162 hours.

Trichloroethylene exhibits slight reproductive toxicity but has apparently no embryotoxic or teratogenic potential. Sperm from mice exposed to 0.3% trichloroethylene vapor 4 h/d for 5 days were reported to have increased abnormalities. No fetal toxicity or teratogenicity was found among offspring of mice or rats exposed to 3000 ppm trichloroethylene 7 h/d on days 6 through 15 of gestation. Anomalies of skeletal and soft tissue indicative of developmental delay were observed among offspring of rats exposed to 1800 ppm 6 h/d from the first through the twentieth day of gestation. Dams showed an increased incidence of displaced right ovary.

The carcinogenic potential of trichloroethylene has been the subject of several investigations. The results of an original investigation showing a dose-related increase in the incidence of hepatocellular carcinomas in mice following oral exposure to trichloroethylene, which had been questioned on methodological grounds, have largely been confirmed. In the original study

B6C3F1 mice exposed by oral gavage to a time weighted average of 2339 or 1 169 mg/kg (males) or 1739 and 869 mg/kg (females) 5 times per week for 78 weeks and sacrificed at 90 weeks showed an increased incidence of hepatocellular carcinomas. Osborne-Mendel rats exposed to 1097 or 549 mg/kg according to the same paradigm and sacrificed at 110 weeks exhibited no increase in tumors. An increased incidence of hepatocellular carcinomas among trichloroethylene exposed B6C3F1 mice was confirmed in a second study in which subjects were exposed to 1000 mg/kg 5 days per week for 103 weeks. Fischer 344 rats exposed to 500 or 1000 mg/kg at the same time failed to show a carcinogenic response.

A modest increase in the incidence of hepatocellular carcinomas has also been observed in B6C3F1 mice, but not Charles River rats, exposed for 2 years, 6 h/d, 5 days per week to 600 ppm trichloroethylene. In another inhalation study, female Han:NMRI mice exposed to 100 or 500 ppm trichloroethylene 6 h/d, 5 days per week for 18 months and sacrificed at 30 months showed an increased incidence of malignant lymphomas. Han:Wist rats and Syrian hamsters exposed according to the same paradigm were unaffected. Results of this last study have been questioned because of a higher than expected incidence of lymphomas in the unexposed mice. Therefore, more inhalation studies involving B6C3F1, Swiss albino, Swiss and ICR/Ha mice and Sprague Dawley rats are under way. An additional gavage study using Osborne-Mendel, Marshall 540, August 28807 and ACI rats is also in progress.

EPA's Carcinogen Assessment Group has estimated a unit risk for trichloroethylene of 1.9×10^{-2} $(mg/kg/d)^{-1}$ based on carcinogenicity data from oral studies in animals (CAG 1984). There is currently no epidemiological evidence which suggests that trichloroethylene is carcinogenic in humans.

A-3 TOXICITY PROFILES FOR METALS

ARSENIC

The toxicity of arsenic compounds varies greatly depending on the chemical form (organic or inorganic) and the oxidation state of arsenic. Trivalent compounds are more toxic than pentavalent compounds.

Long-term inhalation of inorganic arsenic compounds affects the respiratory system in humans (including perforation of the nasal septum). Chronic exposure to arsenic is also associated with skin lesions, peripheral neuropathy, cardiovascular disorders, and peripheral vascular disorders (blackfoot disease) (IARC 1980, USEPA 1980). Smelter workers exposed to arsenic compounds (and possibly other toxic compounds) during pregnancy have had an increased number of infants with low birth weights and increased frequencies of abortion and malformations. Arsenic compounds have also increased the frequency of chromosomal aberrations in humans (IARC 1980, USEPA 1980i).

In several mammalian species, arsenic and arsenate compounds are embryotoxic and/or teratogenic when given orally or by injection (USEPA 1980, IARC 1980). In addition, enzyme systems in a wide variety of species are inhibited by trivalent arsenic compounds (USEPA 1980i). Arsenic compounds induce chromosomal aberrations and morphological changes in mammalian cells.

Inorganic arsenic compounds are classified as human carcinogens by IARC (1980) causing skin cancer in persons exposed to inorganic arsenic through drugs, drinking water, and pesticides and lung cancer in workers exposed to

airborne arsenic compounds. In addition, excess incidences of liver cancer, lymphomas, leukemia, myeloma, and colon cancer in various occupationally-exposed groups occurred (IARC 1980, USEPA 1980). The evidence for carcinogenicity in animals is limited. Increased incidences of leukemia and lymphomas occurred in mice given subcutaneous injections of sodium arsenate during pregnancy and lung tumors developed in rats given a calcium arsenate-copper mixture by intratracheal administration (IARC 1980, USEPA 1980).

The EPA's Carcinogen Assessment Group (CAG) has developed a UCR based upon the studies conducted in Taiwan (Tseng et al. 1968; Tseng 1977) in which an increase in skin cancer was associated with the amount of ingestion of arsenic-contaminated drinking water. The EPA Office of Drinking Water established a Maximum Contaminant Level (MCL) and primary drinking water standard under the Safe Drinking Water Act for arsenic at 0.05 mg/L. In addition, the Safe Drinking Water Committee of the NAS indicated that 0.05 mg/L provides a sufficient margin of safety (NRC 1983). If the CAG risk estimates are applied to the MCL, however, a lifetime cancer risk of ca. 2.5 per 100 is projected. Clearly, EPA cannot have accepted a risk of this magnitude (which is of epidemic proportion) in establishing the MCL. Thus EPA does not appear to view the CAG estimates as realistic. At this time there are no other relevant studies upon which to base an oral UCR for arsenic. Consequently an ADI for ingestion was established as described below.

Mizuta et al. (1956 as cited in NRC 1977) observed that human ingestion of 0.04 mg/kg/d of calcium arsenate resulted in facial edema and peripheral

neuropathy in persons exposed over a 2 to 3 week period. Applying a 100-fold safety factor to this human LOEL results in an ADI of 4.0×10^{-1} μg/kg/d for the ingestion route.

EPA's Carcinogen Assessment Group developed an estimate of excess lung cancer risk associated with arsenic inhalation in workroom area which translates to a unit cancer risk of approximately 12.7 $(mg/kg/d)^{-1}$ (Federal Register 1983).

BERYLLIUM

Human toxicity from exposure to beryllium is primarily a result of the inhalation of beryllium compounds. Acute toxic effects of beryllium inhalation include nasopharyngitis, tracheobronchitis, and in severe cases chemical pneumomitis, which may lead to pulmonary edema and death (Hammond and Beliles, 1980). Contact dermatitis and other dermatological effects (ulceration and granulomas) have been documented in exposed workers. Chronic beryllium disease primarily affects the respiratory system and results in dyspnoea, cough, and weight loss. The respiratory disease is classified as a granulomatosis and may eventually lead to fibrosis, emphysema, and cor-pulmonale. Hypersensitization appears to play an important role in the disease and might result in the development of beryllium disease in individuals with low exposures.

Beryllium appears to have the potential to cause mutations. Positive mutagenic responses have been reported in studies using cultured mammalian cells. Beryllium compounds have also been found to cause chromosomal aberrations in cultured human lymphocytes.

Positive evidence of carcinogenesis has been reported in some mammalian studies. Carcinogenesis has been successfully induced in rabbits by intravenous or intramedullary injection and also in rats when exposed by inhalation or intratracheal injection. Using the IARC criteria requires that beryllium be placed in the "sufficient evidence" category of animal carcinogens (USEPA, 1984).

The evidence for the carcinogenicity of beryllium and beryllium compounds in humans is inconclusive. Early studies (IARC, 1972, 1980; Bayliss et al., 1971; Bayliss and Lainhart, 1972 as cited in USEPA Review Draft, 1984) did not report any positive evidence of carcinogenicity in humans, but more recent studies indicated an increased risk of lung cancer in beryllium-exposed workers (USEPA Review Draft, 1984). The overall animal and human evidence for the carcinogenicity of beryllium results in its placement in a IARC category for chemicals which are "probably carcinogenic in humans" (USEPA Review Draft, 1984).

EPA's Carcinogen Assessment Group estimates a unit risk for beryllium of 2.60 $(mg/kg/d)^{-1}$ based on carcinogenicity data from studies involving human occupational (inhalation) exposure (USEPA, 1985).

CADMIUM

Human toxicity from exposure to cadmium is well-documented. Acute toxic effects of cadmium inhalation include irritation of the upper respiratory tract, chest pains, nausea and diarrhea, dizziness, and death usually due to massive pulmonary edema. Major toxic effects of cadmium ingestion include nausea, vomiting, salivation, diarrhea, abdominal cramps, and in some cases death (Doull et al. 1980). Following chronic exposure to cadmium the principal target organs are the lungs and kidney. Chronic inhalation of cadmium fumes and dust can lead to an emphysema-like condition with loss of ventilatory capacity, increased residual lung volume, and shortness of breath (Doull et al. 1980). The kidney appears to be the most cadmium-sensitive organ, primarily because of its prediliction for accumulation of cadmium. Renal tubular damage is the most important effect of cadmium exposure, irrespective of the route of exposure. The damage is usually characterized by proteinurea; other signs of renal tubular damage may include a reduced ability to concentrate urine, glycosuria, hypercalcinuria, aminoaciduria, and increased uric acid excretion. Excessive exposure to cadmium through the ingestion of contaminated food and drinking water results in a disease characterized by osteomalacia and renal tubular dysfunction (Itai-itai disease).

The toxic effects of cadmium observed in humans also occur in experimental animals. Rats exposed to a cadmium aerosol for 15 days develop lung inflammation followed by emphysema and fibrosis. Signs similar to those of Itai-itai disease develop experimentally in rats given an excess of cadmium in

a calcium deficient diet. In newborn animals, cadmium causes cerebral and cerebellar damage. Cadmium is also toxic to the testes of rats and mice, and causes hyperglycemia and glucose intolerance in animals. Cadmium is teratogenic and fetotoxic in rats, mice, and hamsters (Doull et al. 1980). With regard to mutagenicity, cadmium can cause chromosomal or mitotic aberrations in mammalian cell assays and increase error frequency in DNA synthesis. Mixed results have been obtained for cadmium in microbial mutagenicity assays. Drosophila studies and dominant lethal mutation studies on cadmium were negative (USEPA 1980i).

Several epidemiological studies of workers exposed to cadmium oxide dust provide evidence that cadmium increases the risk of prostatic cancer and respiratory cancer (Stokinger 1981a, USEPA 1980, NAS 1980). IARC (1976) concludes that cadmium and certain cadmium compounds are probably carcinogenic to humans. Animal studies demonstrate that the injection of certain cadmium compounds causes both sarcomas at the site of injection and testicular tumors (Leydig or interstitial cell tumors). Several long-term feeding and inhalation studies have been carried out with cadmium compounds; the induction of tumors by these routes of exposure has not occurred (USEPA 1980j).

EPA's Carcinogen Assessment Group estimates a unit risk for cadmium of 6.6 $(mg/kg/d)^{-1}$, based on carcinogenicity data from studies involving human occupational (inhalation) exposure (CAG 1984).

The National Academy of Sciences has derived a Suggested No-Adverse Level (SNARL) using data from Decker et al. (1958 as cited in NRC 1980). In this study, rats were administered cadmium in drinking water at a concentration

of 10 ppm for one year without effect. The rats consumed an average of 30 mL/d in this study. After exposing rats to 50 ppm cadmium in drinking water for 3 months, effects such as anemia were observed. EPA did not consider cadmium a carcinogen by the oral route when it developed its Ambient Water-Quality Criterion (USEPA 1980j). NAS applied a 1000-fold safety factor to the 10 ppm (or 0.75 mg/kg/d) NOEL to derive the chronic SNARL.

Based upon the Decker Study, and applying a 1000-fold safety factor, an ADI of 7.5×10^{-1} μg/kg/d is recommended for the ingestion route of exposure.

CHROMIUM

Chromium compounds can occur in any of three oxidation states, 0, +3, and +6. The biological properties of chromium compounds vary with the oxidation state. Chromium (VI) is the most hazardous form of chromium.

The pulmonary carcinogenicity of chromium, presumably chromium (VI), is well-documented for humans. Increased incidences of lung cancer are associated with occupational exposure to chromium compounds by inhalation; there is no evidence of increased risk of cancer from ingestion (IARC 1980, USEPA 1980k). In addition, inhalation exposure to elevated concentrations of chromium (VI) compounds causes mucosal irritation, ulceration, and perforation in the respiratory system of humans. Dermal exposure results in primary irritation and allergic contact sensitization in relatively high numbers of exposed individuals (USEPA 1980). An increased incidence of chromosomal aberrations occurred in workers exposed to chromium (VI) compounds (IARC 1980, USEPA 1980k).

Administration of Cr (VI) to experimental animals by various routes causes liver damage, kidney damage (confined to the proximal convoluted tubules), and chronic irritation of the respiratory tract. Some embryotoxic and teratogenic effects developed in mice and hamsters given Cr (VI) at very high doses; it is unclear whether these effects are due to maternal toxicity (IARC 1980). Chromium compounds cause mutations, cell transformations, and chromosomal aberrations in numerous prokaryotic and eukaryotic systems, both *in vivo* and *in vitro* (USEPA 1980k, IARC 1980).

The carcinogenic potential of chromium (VI) in animals either by inhalation or ingestion has not been successfully demonstrated. Tumors in experimental animals have been induced only by implantation of pellets containing chromium (VI) compounds or by injection of chromium (VI) compounds (IARC 1980, USEPA 1980).

EPA's Carcinogen Assessment Group (CAG 1984) estimates a unit risk for chromium of 41 $(mg/kg/d)^{-1}$ based on carcinogenicity data from studies of human occupational exposure by inhalation. For noncarcinogenic endpoints, EPA has estimated an ADI of 2.2×10^{-3} mg/kg/d based on an oral study in rats (USEPA 1984).

COPPER

Copper toxicity produces a metallic taste in the mouth, nausea, vomiting, epigastric pain, diarrhea, and depending on the severity, jaundice, hemolysis, hemoglobinuria, hematuria, and oliguria (USEPA 19801). The toxic intake level for inorganic copper for an adult male is greater than 15 mg/day. Data on effects of chronic exposure to copper come from studies of vineyard workers exposed to copper sulfate and lime mist and copper miners exposed to copper sulfide and oxide dusts. In the former instance, some evidence of copper deposition in the lung and cancer of the lung and liver was found. Copper miners apparently do not show any evidence of an increased body burden of copper (NAS 1977).

Chronic toxicity of copper has been studied in animals and a wide variability in sensitivity to copper has been found, e.g., sheep are very sensitive, rats are very resistant. Copper toxicity in sheep and cattle is characterized by excessive hepatic stores, hemoglobinuria, and hemolysis (NAS 1977). Little data exist concerning the carcinogenicity, mutagenicity, and teratogenicity of copper.

In swine and rats the chronic NOEL in the diet was 250 ppm; applying a safety factor of 100 to this figure would result in a daily dose of 150 μg/kg/d (Boyden et al. 1958; Suttle and Mills 1966a, 1966b; Wallace et al. 1960, all as cited in NRC 1977).

The National Academy of Sciences (NAS), however, (1980 as cited in NRC 1980) indicates that 2 to 3 mg/d (equivalent to 28.6 to 42.9 μg/kg/d) is an adequate and safe intake of copper for adults. These levels are set to assure

a balance between essential and toxic exposure to copper; any intake within the recommended range is considered adequate and safe. The NAS analyses indicate that the EPA drinking water standard of 1 mg/l (28.6 μg/kg/d) would appear adequate to protect the health of persons from toxicity due to copper in drinking water.

Based upon the lower "safe and adequate" limit for adults (NRC 1980), an ADI of 28.6 μg/kg/d is recommended.

LEAD

There are various physiological levels and exposure ranges at which the effects of lead in man occur. The most important effects are those in the hematological, neurobehavioral, and reproductive systems (NAS 1972).

In the hematopoeitic system, anemia is a clinical feature of lead intoxication, resulting from both increased erythrocyte destruction and decreased hemoglobin synthesis (USEPA 1977). In children, who are more sensitive to this effect than adults, a threshold blood lead level for production of this effect is approximately 4 mg/100 mL. Hemoglobin synthesis is impaired by lead which inhibits the synthesis of the globin moiety and several steps in the synthesis of heme. The step most sensitive to lead in the heme synthetic pathway is that mediated by the enzyme δ-aminolevulinic acid dehydratase (δ-ALAD). Inhibition of δ-ALAD occurs at blood lead levels as low as 10 μg/100 mL. Though the significance to human health of δ-ALAD-inhibition at a blood level of 10 μg/100 mL is open to debate, the increased urinary excretion of δ-aminolevulinic acid that occurs with increasing inhibition of 40 μg/100 mL is accepted as a measure of probable physiological impairment in vivo (USEPA 1977). This threshold level for urinary δ-ALAD excretion appears to apply to both adults and children.

An increase in free erythrocyte protoporphyrin (FEP) occurs at blood lead levels of 15 μg/100 mL in children. Elevation of free erythrocyte protoporphyrin has the same implications of physiological impairment in vivo as urinary δ-ALAD. To the extent that the protoporphyrin elevation is a likely indicator of the impairment of mitochondrial function in erythroid tissue, it may be even more important.

The most severe effects of lead poisoning, acute or chronic encephalopathy and/or death, may occur in some adults at blood levels slightly below 100 μg/100 mL (USEPA 1977). For children, encephalopathy occurs at lower levels down to about 80 μg/100 mL. The blood lead levels at which neurobehavioral deficits occur in otherwise asymptomatic children appear to start at a range of 50 to 60 μg/100 mL.

Placental transfer of lead has been demonstrated both by fetal tissue analysis and comparison of newborn umbilical cord blood lead with maternal blood lead. The prenatal exposure of the fetus to lead, even in the absence of teratogenic effects, adversely affects health. Studies have related fetal lead levels to changes in fetal heme synthesis and to the incidence of premature births. Maternal blood lead levels of approximately 30 μg/100 mL may be associated with a higher incidence of premature delivery and premature membrance rupture, but these observation require confirmation (USEPA 1977).

The effects of lead exposure described for humans also occur in a variety of animal species. In addition, carcinogenicity assays have been performed using several lead-containing compounds. Although data exist on animals which have experienced a carcinogenic effect for certain lead salts, no conclusive evidence exists that inorganic lead or organic lead compounds are carcinogenic (IARC 1980). Unfortunately, a paucity of information exists on the teratogenicity and developmental toxicity of chronic low or moderate lead exposures. Other data suggest, however, that chronic low-level lead exposure may induce postnatal developmental delays in rats.

Based on data on blood lead levels associated with hematologic effects in children (Maximum safe blood lead level for children = 30 μg/100 mL), background lead consumption, and correlations between blood lead levels and oral lead intake, USEPA (1980m) estimates that a water concentration of 50 μg/L is an upper limit of acceptability for lead in water. This is equivalent to intake of 1.5 μg/kg/d (assuming 2 liters per day and a 70 kg adult). This is the ADI recommended for the oral and inhalation routes.

MERCURY

Chronic exposure to inorganic mercury produces symptoms which vary widely among individuals. Symptoms may include fatigue, weakness, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, metallic taste in the mouth, increased salivation, soreness of mouth or throat, gum inflammation, black line on the gums, loosening of teeth, irritability, loss of memory, and tremors of the eyelids, lips, tongue, or fingers. Relatively high exposures produce extreme irritability, excitability, delirium with hallucinations, melancholia, anxiety, or manic depressive psychosis. Generally, chronic exposure to mercury produces gingivitis, excess salivation, increased irritability, and muscle tremors (NIOSH 1977).

The principal effects of chronic mercury exposure in laboratory animals are renal damage and nervous system toxicity. Fitzhugh et al. (1950, as reported in Stokinger 1981a) observed granularity, fibrosis, and enlargement of the kidneys, and hypertrophy and dilation of the proximal convoluted tubules in rats chronically fed mercuric acetate. Peripheral nervous system damage occurs in rats (USEPA 1980n). In rats exposed prenatally to mercury vapor, death occurs within 6 days of birth (Baranski and Szymczyk 1983, as reported in USEPA 1980). Gale and Ferm (1971, as reported in USEPA 1980n) observed teratological effects in "animals" exposed to mercuric chloride. In mice treated with mercuric chloride, reversible inhibition of spermatogonial cells developed (Lee and Dixon 1975, as reported in USEPA 1980).

Druckey et al. (1957, as reported in USEPA 1980n) reports that when metallic mercury is injected intraperitonally into rats, sarcomas develop only

in those tissues that are in direct contact with the metal. No other data were found on the potential carcinogenicity of inorganic mercury.

EPA has derived an ADI of 2.8×10^{-4} for mercury. According to USEPA (1980n) it appears that the earliest detected nervous system effects of methyl mercury exposure in man are at blood concentrations of 200-500 ng Hg/mL, which correspond to long-term daily intakes of 200 to 500 mg Hg/70 kg. The 200 µg/d dose corresponds to 2.86 µg/kg/d. In establishing its ambient water quality criterion, EPA applied a 10-fold safety factor to this LOEL for methyl mercury. The EPA drinking water standard (maximum contaminant level) is 2 µg/L, corresponding to a toxicity reference dose of 0.057 µg/kg/d, providing a safety margin of 50 to the LOEL. As all mercury exposure is unlikely to represent methyl mercury, the application of 50-fold safety factor is reasonable and therefore, an ADI of 5.7×10^{-2} µg/kg/d is recommended.

NICKEL

In humans, dermal sensitization is the most common toxic reaction resulting from exposure to nickel and nickel compounds. Dermal exposure may also result in chronic eczema or neurodermatitis. Exposure to nickel and its compounds can lead to irritation of the conjunctiva and the mucous membranes of the respiratory tract (NIOSH 1977). Asthmatic lung disease is documented for nickel plating workers. Cancers of the lung and nasal sinuses occur in nickel refinery workers.

In experimental animals, oral administration of nickel metal is comparatively non-toxic; dogs and cats tolerate up to 12 mg nickel per kg daily for periods up to 200 days without ill effects (Stokinger 1983, as reported in USEPA 1980o). At higher doses acute gastrointestinal irritation with vomiting and diarrhea occurs. By subcutaneous or intravenous injection, gastrointestinal and central nervous system effects, including tremor, chorea-like movements, and paralysis, also arise (Browning 1969).

One researcher found malformations in hamster embryos when the mother was exposed to unidentified nickel compounds administered parenterally at dosages ranging from 0.7 to 10.0 mg/kg (Ferm 1972, as reported in USEPA 1980o). No teratogenic effects occurred when either nickel chloride (16 mg/kg) or nickel subsulfide (80 mg/kg) was administered to rats (Sundermand et al. 1978, as reported in USEPA 1980). Multigenerational reproductive studies have linked oral administration of nickel in the diet to decreases in litter size, and increases in numbers of runts and in neonatal mortality (USEPA 1980o). Male

rats given daily oral doses of nickel sulfate at 25 mg/kg were completely sterile after 120 days (Waltschewa et al. 1972, as reported in USEPA 1980o). Nickel chloride was not mutagenic in a host-mediated assay using NMRI-strain mice. In a study of mutation in a streptomycin-dependent E. coli strain, the nickel compounds, $NiCl_2$, $NiSo_4$, and $Ni(No_3)_2$ were not mutagenic (Stokinger 1981).

A significantly increased incidence of cancers of the lungs and nasal cavities occurred in epidemiological studies of workers in nickel smelters and refineries (NRC 1977). The implicated compounds were nickel subsulfide, nickel oxides, nickel carbonyl vapor, and soluble aerosols of nickel sulfate, nitrate, or chloride (NRC 1980). Tumors developed in rats, mice, guinea pigs, and hamsters administered nickel compounds by inhalation, and by the subcutaneous, intramuscular, intraperitoneal, intratesticular, and intravenous routes. The tumors included adenocarcinomas and squamous cell carcinomas from inhalation, injection-site sarcomas, fibrosarcomas and squamous cell carcinomas from subcutaneous and intratracheal administration, and mesotheliomas from intraperitoneal injection (Stokinger 1981a). EPA's Carcinogen Assessment estimates a unit risk of 1.15 $(mg/kg/d)^{-1}$, based on human occupational (inhalation) exposure (CAG 1984).

The lowest observed effect level for nickel was reported by Schroeder and Mitchener (1971 as reported in NRC 1980). Rats administered 5 ppm nickel in drinking water in a three-generation study showed signs of toxicity including increased perinatal deaths and number of runts; the size of the litter decreased with each generation and few males were born in the third

generation. These findings, however, have not been confirmed by other investigators (NRC 1980). Applying a 1000-fold safety factor to the lowest observed effect level in the Schroeder and Mitchener study would result in a toxicity reference dose of 0.5 μg/kg/d.

Whanger (1973, as cited in Stokinger 1981a) reported that a dose-related reduction in body weight gain occurred when rats were fed at 100, 500, and 1000 ppm nickel acetate in the diet. The application of a 1000-fold safety factor to the 100 ppm lowest observed effect level (in rats) would result in a toxicity reference dose of 6 μg/kg/d.

Nielson (1977 as reported in NRC 1980), indicates that generally 250 μg or more of nickel per gram of diet is required to produce signs of nickel toxicity in rats, mice, chicks, rabbits, and monkeys. Applying a 1000-fold safety factor to this level in rats would result in a toxicity reference dose of 15 μg/kg/d. According to NRC (1980), the daily oral intake averages of nickel by humans range from 165 to 500 μg (2.35-7.14 μ/kg/d); these averages are clearly in excess of the toxicity reference dose which would be derived based on Schroeder and Mitchener (1971) and Whanger 1973.

Based on the available toxicity data and knowledge of actual average human oral intake of nickel which have not been associated with adverse effects, an ADI of 15 μg/kg/d is recommended.

ZINC

Zinc is an essential element and is widely used in a number of therapeutic preparations. Most human and experimental animal data indicate that relatively large doses of zinc can be tolerated for long periods. However, toxic effects occur following acute exposures (USEPA 1980p). The most commonly reported effect of high-level zinc exposure in humans is reversible copper deficiency (USEPA 1980p). Metal fume fever is associated with acute occupational exposure to zinc oxide fumes. No adequate occupational exposure data are available to assign a minimum effective dose for metal fume fever (NIOSH 1975). Zinc poisonings in humans also result in nausea, vomiting, fever, anemia, and lethargy; these symptoms are reversible upon cessation of exposure (USEPA 1980p).

Rats exposed to zinc sulfate in drinking water for 14 months showed histopathological changes of several endocrine organs including hypertrophy of the adrenal cortex and changes in the pancreatic islets and pituitary gland (Aughey et al. 1977, as reported in USEPA, 1980p). Drinker et al. (1927, as reported in Stokinger 1981a) observed glycosuria in dogs and fibrous degeneration of the pancreas in cats administered 175 to 1000 mg zinc oxide per day in the diet. Sutton and Nelson (1937, as reported in Stokinger 1981a) reported that greater than 0.05 percent zinc in the diet of rats reduced their capacity to reproduce, and that 1 percent inhibited growth and caused severe anemia.

The zinc-containing fungicides Zineb [zinc ethylenebis (dithiocarbamate)] and Ziram [zinc dimethyl dithiocarbamate] were tested for teratogenic, reproductive, and mutagenic effects. At 50 mg/kg/d, the fungicides retarded

the onset of pregnancy, and produced infertility, fetal resorption, and developmental abnormalities in rats. Zineb and Ziram were not mutagenic in a short-term assay with Drosophila (Ryazanova 1967, as reported in Stokinger 1981a). These positive results may not be relevant to zinc or other zinc compounds. No data were found on the carcinogenic potential of zinc.

Because zinc is an essential nutrient for humans, a recommended dietary allowance of 15 mg/d in adults has been established (NAS 1980 as reported in NRC 1980). This corresponds to 214 μg/kg/d assuming a 70 kg adult. An ADI of 2.1×10^{-1} mg/kg/d is therefore recommended for the oral and inhalation routes.

A-4 TOXICITY PROFILES FOR ACID GASES

HYDROGEN CHLORIDE

A wide variety of effects has been reported to be associated with exposure to hydrogen chloride, including necrosis of the tracheal and bronchial epithelia, pulmonary edema, atelectasis, emphysema, damage to the pulmonary blood vessels, liver lesions, nausea, and vomiting (Stokinger 1981b; NRC 1976). Henderson and Haggard (1943 as cited in ACGIH 1981) reported that exposure of men to 50-100 ppm HCl for one hour was barely tolerable, and 35 ppm caused irritation of the throat after a "short" exposure; 10 ppm was the maximum concentration tolerated for prolonged exposure. In workers exposed occupationally to concentrations of less than 1 ppm HCl, no statistically significant responses were seen (Patil et al. 1970 as cited in NRC 1976).

ASEL

ACGIH has set a ceiling limit of 5 ppm; this concentration should not be exceeded even instantaneously in occupational settings (ACGIH 1981). To estimate an acute NOEL for hydrogen chloride a safety factor of 10, to account for sensitive segments of a population, was applied to the concentration of 35 ppm (corresponding to LOEL in an exposed populations) and a safety factor of 2 was applied to convert the LOEL to a NOEL. This resulted in an acute safe exposure level (ASEL) of 1.75 ppm or 2.6 mg/m^3.

STPL

The National Academy of Sciences (1971) reviewed HCl and established guides for short-term exposure of the public. They conclude that the chief effect of inhalation exposure to HCl will be primary irritation of the moist mucous membranes of the upper respiratory tract. Prevention of the disagreeable and

irritating odor of HCl was thus used as the basis for establishing short-term public exposure limits (STPL's). They assumed that greater than 5-10 ppm is disagreeable and irritating and should be avoided. The lowest concentration that could be smelled was between 1 and 10 ppm. The following STPL's were established by NAS and will be used for this report.

Short-Term Public Limits (STPL's)

10 min	6 mg/m^3 (4 ppm)
30 min	3 mg/m^3 (2 ppm)
60 min	3 mg/m^3 (2 ppm)
1 h daily	1 mg/m^3 (0.7 ppm)

The NAS (1971) emphasized that these levels are time-weighted averages not considered to present any health hazard. It should be recognized that excursions above these levels are likely to produce objectionable odors and/or irritation.

HYDROGEN CYANIDE

A considerable amount of information has been reported regarding human exposure to hydrogen cyanide. Four basic categories of effects result from acute exposure to cyanide: hematological, cardiovascular, neurological and metabolic (USEPA 1981a). The LC_{50} for a 30 second exposure ranges from 450 mg/m^3 in the mouse to 2500 mg/m^3 in the rat (McNamara, 1976 as cited in USEPA 1981a). Symptoms of exposure in workers exposed occupationally to cyanide include headache, weakness, change in taste and smell, irritation of throat, vomiting, abdominal colic, pericardial pain and nervous instability (ACGIH 1981).

Patty (1963 as cited in ACGIH 1981) compiled the following table regarding human response to acute hydrogen cyanide exposure:

Response	Concentrations mg/m^3
Immediate fatal	300
Fatal after 10 min	200
Fatal after 30 min	150
Fatal after ½ to 1 h or later or dangerous to life	120-150
Tolerated for ½ to 1 h without immediate or late effects	50-60
Slight symptoms after several hours	20-40

ASEL

The acute NOEL is estimated by applying safety factors of 10 for sensitive segments of the population, and 2 for conversion of a LOEL to a NOEL based on the occurrence of "slight symptoms" upon exposure to a concentration of 20 mg/m^3 for several hours. This calculation yields a recommended (ENVIRON) acute safe exposure level (ASEL) of 1.0 mg/m^3.

HYDROGEN FLUORIDE

Exposure to high concentrations of hydrogen flouride results in a variety of symptoms. When guinea pigs and rabbits were exposed to concentrations of approximately 50 mg/m^3 symptoms observed included signs of mild irritation, such as coughing and sneezing. At higher concentrations, signs of more severe irritation were observed, such as closing of the eyes, paroxysms of coughing and sneezing, slowing of respiration and discharge from nose and eyes (Machle et al, 1934 as cited in Stokinger 1981b). Rats were more sensitive to HF toxicity than rabbits, guinea pigs and dogs (Rosenholz et al, 1963 as cited in Stokinger 1981b). The LC_{50} for the rat is 4060 mg/m^3 for a 5 minute exposure, 1670 mg/m^3 for a 30 minute exposure and 1070 mg/m^3 for a 60 minute exposure. Signs of toxicity apart from irritation included pathological lesions in the liver and kidney (Rosenholz et al, 1963, as cited in Stokinger 1981b).

There is less information regarding acute exposure of humans to hydrogen flouride. The highest concentration tolerated for one minute was reported to be 98 mg/m^3. Symptoms at this concentration included conjunctival and respiratory irritation and smarting of the skin. At 26 mg/m^3 there was a mild smarting of the nose and eyes (Patty 1949 as cited in ACGIH 1981). Slightly longer exposure of humans, 6 h/d for 10 to 50 days, resulted in some irritation of nose and eyes at concentrations above 3 ppm. (Largent 1961 cited in ACGIH 1981), and ACGIH has set a TLV of 2.5 mg/m^3 (equivalent to about 3 ppm) hydrogen flouride (as F) for occupational exposure during a

normal working week (ACGIH 1981). Applying a safety factor of 10 to account for sensitive segments of the population and, a safety factor of 2 to convert the acute LOEL of 26 mg/m^3 to a NOEL, based on mild irritation to the nose and eyes, results in an acute safe exposure level (ASEL) of 1.3 mg/m^3.

HYDROGEN SULFIDE

Acute exposure to hydrogen sulfide results predominantly in respiratory irritation. At extremely high concentrations (500-1000 ppm) unconsciousness and death may occur through respiratory paralysis (ACGIH 1981). Other symptoms of hydrogen sulfide exposure include CNS symptoms such as nervousness, cough, nausea, headache and insomnia as well as eye irritation (ACGIH 1981). The LC_{50} in mice is 673 ppm for a one hour exposure (NIOSH 1983).

The National Research Council (NRC 1979) compiled a table of human responses to various acute concentrations of hydrogen sulfide:

Effect	Concentration (mg/m^3)
Immediate collapse with respiratory paralysis	1400-2800
Strong nervous system stimulation, apnea	700-1400
Pulmonary edema	420-700
Olfactory paralysis	210-350
Threshold of serious eye injury	70-140
Offensive odor	4.2-7
Approximate threshold for odor	0.14-0.28

ACGIH has set a TLV for occupational exposure to hydrogen sulfide during a normal work week of 14 mg/m^3 (ACGIH 1981) based on the occurrence of headache, sleep disturbances, nausea, weight loss. Based on the threshold for

eye injury of 70 mg/m^3 and applying a safety factor of 10 to protect sensitive segments of the population and 10 for the conversion of a LOEL to a NOEL for a serious effect, result in an estimated acute safe exposure level (ASEL) of 0.7 mg/m^3.

NITROGEN DIOXIDE

Exposure to nitrogen dioxide results in respiratory effects such as decreased lung capacity and bronchoconstriction. Other reported symptoms include a decreased resistance to disease and polycythemia (WHO 1977). The human olfactory threshold is 240 mg/m^3 while functional changes in the lungs of healthy human subjects begin after 10 minutes exposure to concentrations of 1300 mg/m^3 (WHO 1977). Increased reaction to a bronchoconstrictor was seen in asthmatic subjects after exposure to NO_2 concentrations of 190 mg/m^3 for one hour (Orehek et al. 1976 as cited in WHO 1977). Concentrations of 20 to 40 mg/m^3 have been reported to irritate the eyes, nose, and the respiratory tract (Patty 1949 as cited in ACGIH 1983).

Adley (1946 cited in ACGIH 1981) reported that "short" exposures to NO_2 averaging 25-38 ppm resulted in no observable effects and Norwood et al. (1966 cited in ACGIH) reported that 90 ppm for 30 min resulted in pulmonary edema. The USEPA (1982) summarized data showing that acute human exposures to nitrogen dioxide concentrations of approximately 300 ppm result in death; acute exposure to 150-200 ppm results in severe respiratory distress; acute exposures to 50-100 ppm result in reversible bronchiolitis; and exposures to 25-75 ppm result in reversible bronchial injury. Increased airway resistance was observed in healthy adults with a single 2-h NO_2 exposure of from 2.5-7.0 ppm, and altered airway resistance has been reported among healthy subjects exposed to concentrations somewhere in the range from 0.7-2.0 ppm (Suzuki and I Shikawa 1965 cited in USEPA 1982).

If two ppm of NO_2 (equivalent to about 4 mg/m^3) is considered a LOEL, a safety factor of 2 can be used to convert a LOEL of 4 mg/m^3 from the above study to a NOEL. Applying a safety factor of 10, to account for sensitive components of the population results in an acute safe exposure level (ASEL) of 0.2 mg/m^3.

SULFUR DIOXIDE

Sulfur dioxide is a strong irritant which will primarily affect the mucous membranes of the respiratory tract and the conjuctiva of the eye. Sulfur dioxide is very soluble in water and forms sulfuric acid in solution. The mucous present in nasal cavities can absorb large amounts of SO_2, therefore, the response to sulfur dioxide is more severe when inhaled through the mouth rather than the nose. The high water solubility also causes the upper respiratory tract to respond immediately to sulfur dioxide's local irritant properties through bronchoconstriction. Therefore, people with pre-existing respiratory problems, such as asthmatics and the elderly, are at greater risk of adverse effects of sulfur dioxide exposure (ACGIH 1981; Stoker and Seager 1976). Other symptoms associated with acute exposure to sulfur dioxide include excess salivation, moderate dyspnea, lacrimation, conjunctivitis, abdominal distension and lethargy (EPRI 1976).

Exposure of human subjects to 1 ppm of sulfur dioxide for 10 min caused a slight increase in lung air-flow resistance in only one of 11 subjects. Raising the concentration to 5 ppm caused a 72% increase (Frank et al., 1962). This response was not dose dependent but concentration dependent because increasing exposure period to 30 min did not cause an increase in the effect. A study by Tomono 1961 (as reported by ACGIH 1981) reported 1.6 ppm exposure was the lowest dose which produced bronchoconstriction in healthy subjects.

Stoker and Seager (1976) have summarized the effects of short-term SO_2 exposure on humans:

Effect	Concentration ppm
Immediate coughing	20
Eye irritation	10
Immediate throat irritation	8-12
Threshold for inducing reversible bronchial constriction in unimpaired individuals	1.6
Threshold for odor recognition	0.5
Threshold for taste recognition	0.3

ASEL

EPRI (1976) noted that in healthy males exposed to sulfur dioxide, 1 ppm is considered the threshold for bronchoconstrictor effects. A clear trend in increased pulmonary resistance is not seen until exposure concentrations reach several ppm. ACGIH has set a Threshold Limit Value of 2 ppm for occupational exposure to sulfur dioxide during a normal work week (ACGIH 1981). Using 1 ppm as the threshold for effects due to sulfur dioxide and applying a safety factor of 10 to protect sensitive segments of the population, the recommended acute safe exposure level (ASEL) is 0.1 ppm (0.26 mg/m^3).

STPL

NAS (1984) reviewed the data available on sulfur dioxide exposure and emphasized that changes in airway resistance are shortlived (about 5 min) at exposure levels of 30 ppm or less in (Attaway and Tyler 1976; Lawther et al.

1975 both as cited in NAS 1984). Although recovery of normal bronchial functioning is rapid in normal resting subjects, it may take 30-60 minutes after exercise, for asthmatics (Bates and Hazucha, 1973; Koenig et al., 1981; Gokemeijer et al. 1973; Lawther et al. 1975; all as cited in EPA 1982). The NAS (1984) estimated that 10-20% of the population will respond with bronchial hyperreactivity. OSHA (1983) recommended a TWA limit of 2 ppm. ACGIH (1980, 1983) recommended a TLV-TWA of 2 ppm for an 8-h period and a STEL of 5 ppm for 15 min. The reduction of the TLV from 5 to 2 ppm was based on available data and, in the opinion of the ACGIH committee, effects reported below 2 ppm were not serious enough to justify a lower limit.

NAS (1984) established emergency exposure limits (EELs) and continuous exposure limits (CELs) for sulfur dioxide. Emergency exposure limits relate to unpredictable single exposures, usually lasting 60 minutes or less, but never more than 24 hours, and which occur rarely in a person's lifetime. They reflect the acceptance of the occurrence of a nonincapacitating decrement in performance during emergencies. These EELs are established for defined occupational groups such as military personnel or space personnel, rather than the general public. Based on the reversibility of bronchoconstriction seen at levels below 30 ppm the NAS (1984) established the following EELs in 1984:

10-min EEL	30 ppm
30-min EEL	20 ppm
60-min EEL	10 ppm
24-h EEL	5 ppm

Because the population at risk in an occupational setting is generally younger and healthier than the normal population, short-term public exposure limits (STPLs) can differ somewhat from EELs. We therefore propose that the EELs established by NAS (1984) should be divided by a ten-fold safety factor to establish an STPLs. This would provide an adequate margin of safety for asthmatics and other respiratory compromised individuals. Our STPLs, for sulfur dioxide are listed below:

10-min STPL	3 ppm
30-min STPL	2 ppm
60-min STPL	1 ppm
24-hr STPL	0.5 ppm

REFERENCES

Altman, P.L., and Dittmer, D.S., eds. 1972. Biology Data Book, Second Edition. Federation of American Societies for Experimental Biology, Bethesda, Md.

Aviado, D.M. 1977. Preclinical pharamacology of toxicology and halogenated solvents and propellants. National Institute Drug Abuse Res. Monogr. Ser. 15:165-184.

American Conference of Governmental Industrial Hygienists (ACGIH). 1981. Documentation of the Threshold Limit Values. Fourth Edition. ACGIH, Cincinnati, Ohio

Bowman, R. Heironimus, M., and Barsotti, D. 1981. Locomotor hyperactivity in PCB-exposed Rhesus monkeys. Neurotoxicity 2:251-258

Buser, H.R. 1985. Formation, occurrence, and analysis of polyclorinated dibenzofurans, dioxins and related compounds. Env. Health Persp. 60:259-267.

Carcinogen Assessment Group (CAG). 1984. Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group as Suspect Human Carcinogens. From Draft EPA Health Assessment Document for Carbon Tetrachloride. September 1984

Carlsson, A. and Lindquist, T. 1977. as cited in: Health Assessment Document for Toluene. (Part 2). USEPA, 1982. EPA-600/8-82-008.

Clayton, G.D. and Clayton, F.E. 1981. Patty's Industrial Hygiene and Toxicology. Volume 2A. (John Wiley and Sons).

Clayton, G.D. and Clayton, F.E. 1981. Patty's Industrial Hygiene and Toxicology. Volume 2B. (John Wiley and Sons)

Clayton, G.D. and Clayton, F.E. 1982. Patty's Industrial Hygiene and Toxicology. Volume 2C. (John Wiley and Sons).

Cornish, H.H. 1980. Solvents and Vapors. In Toxicology, The Basic Science of Poisons, Second Edition (J. Doull, C. Klaasen and M. Amdur, eds.), Macmillan Publishing Co., New York

Deichmann, W.B. 1981. Halogenated cyclic hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition (Clayton, G.D. and F.E. Clayton, eds.) Volume 2B, John Wiley and Sons, New York

Deichmann, W.B. and Keplinger, M.L. 1981. Phenols and phenolic compounds. In Patty's Industrial Hygiene and Toxicology, Third Edition (Clayton, G.D. and F.E. Clayton eds.) Volume 2A, John Wiley and Sons, New York

Doull, J., Klaasen, C.D., and Amdur, M.O. 1980. Casarett and Doull's Toxicology. Second Edition. MacMillan Publishing Co., Inc., New York

Electric Power Research Institute (EPRI). 1976. Sulfur Dioxides: Current Status of Knowledge EPRI EA-316. Palo Alto, California

Encyclopedia of Occupational Safety and Health (EOSH). 1971. Volume II: L-Z. International Labour Office. Geneva

Federal Register. 1983. Vol. 48 No. 10 p. 1884, January 14, 1983

Goldstein, J.A., Friesen, M., Linder, R.E., Hickman, P., Hass, J.R., and Bergman, H. 1977. Effects of pentachlorophenol on hepatic drug-metabolizing enzymes and porphyria related to contamination with chlorinated dibenzo-p-dioxins and dibenzofurans. Biochem. Pharm. 26:1549-1557.

Hammond, P.B., and Beliles, R.P. 1980. Metals. In: Doull, J., Klassen, C.D., and Amdus, M.O. eds. Toxicology: The Basic Science of Poisons. Macmillan Publishing Co., New York. pp. 409-467

Heller, V.G., and Purcell, L. 1938. Phenol-contaminated waters and their physiological action. J. Pharmacol. Exp. There. 63:99-107

Hoeltge, Inc. No date. Animal Care Equipment Catalogue. Cincinnati, Ohio.

International Agency for Research on Cancer (IARC). 1973. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 2: Some Inorganic and Organometallic Compounds. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1974a. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 4: Some Aromatic Amines, Hydrazine and Related Substances, N-Nitroso Compounds and Miscellaneous Alkylating Agents. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1974b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Vol. 7: Some Anti-Thyroid and Related Substances, Nitrofurans and Industrial Chemicals. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1976. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 11: Cadmium, Nickel, Some Epoxides, Miscellaneous Industrial Chemicals and General Considerations on Volatile Anaesthetics. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1977. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Volume 15: Some Fumigants, the Herbicides 2,4-D and 2,4,5-T, Chlorinated Dibenzo-dioxins and Miscellaneous Industrial Chemicals. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1979a. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 19: Some Monomers, Plastics and Synthetic Elastomers and Acrolein. World Health Organization, Lyon, France

International Agency for Research on Cancer (IARC). 1979b. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 20: Some Halogenated Hydrocarbons. World Health Organizations, Lyon, France

International Agency for Research on Cancer (IARC). 1980. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 23: Some Metals and Metallic Compounds. World Health Organization, Lyon, France

International Programmer on Chemical Safety (IPCS). 1984. Environmental. Health Criteria 32. Methylene Chloride. World Health Organization. Lyon, France

Kashimoto, T., Miyata, H., Fukushima, S., Kunita, N., Ohi, G. and Tung, T.C. 1985. PCBs, PCQs, and PCDFs in blood of Yusho and Yu-Cheng patients. Env. Health Persp. 59:73-78.

Kimbrough, R.D., Squire, R.A., Linder, R.E., Strandberg, J.D., Montali, R.J., and Burse, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. JNCI 55:1453-1456

Krasavage, W.J., O'Donoghue, J.L. and DiVincenzo, G.D. 1982. Ketones. In Patty's Industrial Hygiene and Toxicology, Third Edition (G.D. Clayton and F.E. Clayton, eds.) Volume 2C, John Wiley and Sons, New York

Lehman, A.J. 1959. Appraisal of the Safety of Chemicals in Foods, Drugs and Cosmetics. Association of Food and Drug Officials of the U.S.

Luster, M.I., Faith, R.E. and Lawson, L.D. 1979. Effects of 2,3,7,8-Tetrachlorodibenzofuran (TCDF) on the immune system in guinea pigs. Drug and Chem. Tox. 2(1 & 2):49-60.

Masuda, Y., Kuroki, H., Haraguchi, K., and Naagayama, J. 1985. PCB and PCDF congeners in the blood and tissues of Yusho and Yu-Cheng patients. Env. Health Persp. 59:53-58.

McNulty, W.P. 1985. Toxicity and fetotoxicity of TCDD, TCDF, and PCB isomers in rhesus macaques (Macaca mulatta). Env. Health Persp. 60:77-88.

Miyata, H., Fukushima, S., Kashimoto, T., and Kunita, N. 1985. PCBs, PCQs, and PCDFs in tissues of Yusho and Yu-Cheng patients. Env. Health Persp. 59:67-72.

Moore, H.R. 1979. Comparative toxicity of three halogenated dibenzofurans in guinea pigs, mice, and rhesus monkeys, In: Annals of The New York Academy of Science: Health Effects of Halogenated Aromatic Hydrocarbons. vol. 320. Nicholson and Moore, Eds. N.Y. Academy of Sciences. New York. Pp. 151-163.

National Academy of Sciences 1971. Guides for Short Term Exposures of the Public to Air Pollutants II. Guide for Hydrogen Chloride. Committee on Toxicology. National Technical Information Service APTD-0764

National Academy of Sciences (NAS). 1972. Lead: Airborne Lead in Perspective. Committee on the Biologic Effects of Atmospheric Pollutants, NAS, Washington, D.C.

National Academy of Sciences (NAS). 1977. Copper. Committee on Medical and Biologic Effects of Environmental Pollutants, Division of Medical Sciences, Assembly of Life Sciences, National Research Council, Washington, D.C.

National Academy of Sciences 1984, Emergency and Continuous Exposure Limits for Selected Airborne Contaminants Vol. 2. Committee on Toxicology. National Academy Press. Washington, D.C.

National Institute for Occupational Safety and Health (NIOSH). 1975. Criteria for a Recommended Standard: Occupational Exposure to Zinc Oxide. DHEW Publication No. 76-104

National Institute for Occupational Safety and Health (NIOSH). 1976. Criteria for a Recommended Standard -- Occupational Exposure to Methylene Chloride. PB81-117017.

National Institute for Occupational Safety and Health (NIOSH). 1976. Criteria for a Recommended Standard: Occupational Exposure to Isopropyl Alcohol. DHEW Publication No. 76-142

National Institute for Occupational Safety and Health (NIOSH). 1977. Occupational Disease: A Guide to Their Recognition. U.S. Department of H.E.W., P.H.S., C.D.C., NIOSH. DHEW (NIOSH) Publication No. 77-181

National Institute for Occupational Safety and Health (NIOSH). Criteria for a recommended standard... Occupational exposure to refined petroleum solvents. DHEW (NIOSH) Publication No. 77-192. 1977

National Institute for Occupational Safety and Health (NIOSH). 1980. Registry of Toxic Effects of Chemical Substances. 1979 Edition. Volume 1. DHHS (NIOSH) Publication No. 80-111.

National Institute for Occupational Safety and Health (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances (RTECS). 1981-82 Ed. Volumes 1-3. DHHS (NIOSH) Publication No. 83-107

National Research Council (NRC) 1976. Medical and Biological Effects of Environmental Pollutants. Chlorine and Hydrogen Chloride. National Academy of Sciences, Washington, D.C.

National Research Council (NRC). 1977. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 1. National Academy Press, Washington, D.C.

National Research Council (NRC) 1979. Medical and Biological Effects of Environmental Pollutants. Hydrogen Sulfide. University Park Press. Baltimore

National Research Council 1979a. Criteria for Short Term Exposures to Air Pollutants. National Academy of Sciences Committee on Toxicology. Washington, D.C.

National Research Council (NRC). 1980. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 3. National Academy Press, Washington, D.C.

National Research Council (NRC). 1981. Formaldehyde and Other Aldehydes. Committee on Aldehydes, Board of Toxicology and Environmental Health Hazards, Assembly of Life Sciences

National Research Council (NRC). 1983. Board on Toxicology and Environmental Health Hazards. Safe Drinking Water Committee. Drinking Water and Health. Volume 5. National Academy Press, Washington, D.C.

National Research Council (NRC). 1983. Drinking Water and Health. Volume 5. National Academy of Sciences, Washington, D.C.

National Toxicology Program (NTP). 1984a. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Benzene (CAS No. 71-43-2) in F344/N Rats and B6C3F1 Mice (Gavage Studies). Draft Report. NTP-TR-289. NIH Publication No. 84-2545. NTP-84-072

National Toxicology Program (NTP). 1984b. Management Status Report. October 24, 1984

Poland, A., Knutson, J., and Glover, E. 1983. A consideration of the mechanism of action of 2,3,7,8-TCDD and related halogenated aromatic hydrocarbons. In: Human and Environmental Risk of Chlorinated Dioxins and Related Compounds. Tucker, Young, and Gray, eds. Plenum Press, New York.

Ontario Ministry of the Environment (OMOE). 1985. Polychlorinated dibenzo-p-dioxin [TCDDs] and polychlorinated dibenzo furans [PCDFs]. Scientific Criteria Document for Standard Development No. 4-84. Prepared for Intergovernmental Relations and Hazardous Contaminants Branch.

Ontario Ministry of the Environment (OMOE). 1984. Preliminary Hearing Document on Proposals for the Regulation of Mobile PCB Destruction Facilities in Ontario. Prepared for presentation to the Commission of Inquiry on the Regulatory Control of Mobile PCB Destruction Facilities. Pp. 37-39.

Reggiani, G. and Bruppacher, R. 1985. Symptoms signs and findings in humans exposed to PCBs and their derivatives. Env. Health Persp. 60:225-232.

Reinhardt, C.F. and Brittelli, M.R. 1981. Heterocyclic and Miscellaneous Nitrogen Compounds. In Patty's Industrial Hygiene and Toxicology, Third Edition, (G.D. Clayton and F.E. Clayton eds.) Volume 2A, John Wiley and Sons, New York

Rowe, V.K. and McCollister, S.B. 1982. Alcohols. In Patty's Industrial Hygiene and Toxicology, Third Edition, (G.D. Clayton and F.E. Clayton, eds.) Volume 2C, John Wiley and Sons, New York

Sandmeyer, E.E. 1981. Aromatic Hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition (G.D. Clayton and F.E. Clayton, eds.) Volume 2B, John Wiley and Sons, New York

Stoker, H.S. and Seager, S.L. 1976. Environmental Chemistry: Air and Water Pollution. Second Edition. Scott Foresman and Company. Glenview, Illinois

Stokinger, H.E. 1981a. The Metals. In Patty's Industrial Hygiene and Toxicology, Third Revised Edition, (Clayton, G.D. and Clayton F.E. eds.) Volume 2A, John Wiley and Sons, New York

Stokinger, H.E. 1981b. The Halogens and the Nonmetals Boron and Silicon. In Patty's Industrial Hygiene and Toxicology, Third Edition, (Clayton, G.D. and Clayton, F.E., eds.) Volume 2B, John Wiley and Sons, New York

Tiesinger, J. 1948. J. Ind. Hyg. Tox. 30:58

Torkelson, T.R. and Rowe, V.K. 1981. Halogenated aliphatic hydrocarbons. In Patty's Industrial Hygiene and Toxicology, Third Edition, (Clayton, G.D. and F.E. Clayton, eds.) volume 2B, John Wiley and Sons, New York

Tseng, W.P. 1977. Effects and Dose-response Relationships of Skin Cancer and Blackfoot Disease with Arsentic. Environ. Health Perspect. 19:109-119.

Tseng, W.P., Chu, H.M., How, S.W., Fong, J.M., Lin, C.S., and Yeh, S. 1968. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

U.S. Environmental Protection Agency (USEPA). 1977. Air Quality Criteria for Lead. Office of Research and Development, USEPA, Washington, D.C. PB-280-411

U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria Documents, Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. (e.g., Ambient Water Quality Criteria for Phthalate Esters, EPA/440/5-80-067. PB81-117780)

U.S. Environmental Protection Agency (USEPA). 1980a. Ambient Water Quality Criteria for Acrylonitrile. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-017. PB81-117285

U.S. Environmental Protection Agency (USEPA). 1980b. Ambient Water Quality Criteria for Benzene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-018. PB81-117293

U.S. Environmental Protection Agency (USEPA). 1980c. Ambient Water Quality Criteria for Chloroform. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-057. PB 81-117681

U.S. Environmental Protection Agency (USEPA). 1980d. Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. Draft report. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1980e. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards, Criteria and Standards, Division, Washington, D.C. EPA 440/5-80-068. PB81-117798.

U.S. Environmental Protection Agency (USEPA). 1980f. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-066. PB81-117772

U.S. Environmental Protection Agency (USEPA). 1980g. (revised 1983). An Exposure and Risk Assessment for Tetrachloroethylene. Final Draft Report

U.S. Environmental Protection Agency (USEPA). 1980h. Ambient Water Quality Criteria for Toluene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-75. PB81-117855

U.S. Environmental Protection Agency (USEPA). 1980i. Ambient Water Quality Criteria for Arsenic. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-021. PB81-117327

U.S. Environmental Protection Agency (USEPA). 1980j. Ambient Water Quality Criteria for Cadmium. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-025. PB81-117368

U.S. Environmental Protection Agency (USEPA). 1980k. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-035. PB81-117467

U.S. Environmental Protection Agency (USEPA). 1980l. Ambient Water Quality Criteria for Copper. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-036. PB81-117475

U.S. Environmental Protection Agency (USEPA). 1980m. Ambient Water Quality Criteria for Lead. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-057. PB81-117681

U.S. Environmental Protection Agency (USEPA). 1980n. Ambient Water Quality Criteria for Mercury. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-058

U.S. Environmental Protection Agency (USEPA). 1980o. Ambient Water Quality Criteria for Nickel. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-080. PB81-117715

U.S. Environmental Protection Agency (USEPA). 1980p. Ambient Water Quality Criteria for Zinc. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA 440/5-80-079. PB81-117897

U.S. Environmental Protection Agency (USEPA). 1981. Advisory Opinion for Dichloromethane (Methylene Chloride), Office of Drinking Water, Draft.

U.S. Environmental Protection Agency (USEPA). 1981a. An Exposure and Risk Assessment for Cyanide. Office of Water Regulations and Standards, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1981b. An Exposure and Risk Assessment for Trichloroethylene. Final Draft Report, March 1981

U.S. Environmental Protection Agency (USEPA). 1981c. An Exposure and Risk Assessment for Nickel. Final Draft Report. U.S. EPA Contract 68-01-5949, Task 3. Office of Water Regulations and Standards (WH-553), Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1981d. An Exposure and Risk Assessment for Arsenic (Final Draft Report). Office of Water Regulations and Standards, Monitoring and Data Support Divisions, Washington, D.C.

U.S. Environmental Protection Agency (USEPA). 1982. Health Effects Assessment Summary for 300 Hazardous Organic Constituents in Support of Regulatory Impact Analysis of the Land Disposal Branch (LDB) and Interim Final Incinerator Regulations of the Technical Branch (TB) of the Office of Solid Wastes, OSWER/EPA, Draft, September 20, 1982

U.S. Environmental Protection Agency (USEPA). 1983. Health Assessment Document for Acrylonitrile. Final Report. EPA-600/8-22-007F.

U.S. Environmental Protection Agency (USEPA). 1983. Health Assessment Document for Dichloromethane (Methylene Chloride). Office of Health and Environmental Assessment, Washington, D.C. December 1983, External Review Draft, EPA-600/8-82-004B.

U.S. Protection Agency. 1984. Research and Development, Summary of Current Acceptable Daily Intakes (ADIs) for Oral Exposure. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

U.S. Environmental Protection Agency (USEPA). 1984. Summary of Current Acceptable Daily Intakes (ADIs) of Oral Exposure. Internal Review Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. February 1984

U.S. Environmental Protection Agency (USEPA). 1984. Air Quality Criteria for Lead. Review draft. Environmental Criteria and Assessment office. EPA-600/8-83-028B

U.S. Environmental Protection Agency (USEPA). 1984. Health Assessment Document for Chromium. Final Report. Publication No. EPA-600-8/83-014F. August, 1984

U.S. Environmental Protection Agency (USEPA). 1984. Health Assessment Document for Beryllium. Review Draft. EPA-600/8-84-026A

U.S. Environmental Protection Agency (USEPA). 1985. Health Assessment Document for Chloroform. Review Draft. EPA-600/8-84-004F

U.S. Environmental Protection Agency (USEPA). 1985. Health Assessment Document for Dichloromethane (Methylene Chloride)

Van Den Berg, M., Olie, K., and Hutzinger, O. 1985. Polychlorinated Dibenzofurans (PCDFs). Tox. Env. Chem. 9:171-217.

World Health Organization (WHO). 1977. Environmental Health Criteria. Oxides of Nitrogen. World Health Organization. Geneva

World Health Organization (WHO). 1983. Styrene, IPCS International Program on Chemical Safety. Environmental Health Criteria 26. World Health Organization, Geneva. p.32.

APPENDIX B

SELECTION OF SURROGATE CHEMICALS

C O N T E N T S

		Page
B.1	INTRODUCTION	B-1
B.2	BASIS FOR HAZARD INDEX	B-6
	Release Potential	B-8
	Toxicity	B-10
	Quantity	B-14
B.3	RESULTS OF SURROGATE SELECTION	B-15
	Selection of Surrogate Chemicals	B-15
	Calculation of Mass Fractions	B-22
B.4	LIMITATIONS OF SURROGATE SELECTION PROCEDURE	B-24
REFERENCES		B-28

LIST OF TABLES

		Page
B-1	OWMC Waste Classes	B-2
B-2	Waste Categories Included in the Organic Constituent Selection Procedure	B-3
B-3	Concentrations of Organic Contaminants in Waste Oils	B-5
B-4	Basis for Hazard Indices	B-7
B-5	Vapor Pressure Scores	B-9
B-6	Acute Toxicity Scores	B-11
B-7	Chronic Toxicity Scores	B-13
B-8	Quantity Scores	B-15
B-9	Environmental Parameters and Associated Scores	B-16
B-10	Basis for Chronic Toxicity Scores	B-17
B-11	Chemicals Scoring the Highest Hazard Index	B-20
B-12	Mass Fractions for Fugitive and Incinerator Emissions	B-23
B-13	Mass Fractions for Truck Spills and Tank Farm Fires	B-25
B-14	Summary of Usable Data Points from Canviro Data Base	B-27

B.1 INTRODUCTION

The surrogate selection procedure was conducted to identify hazardous waste constituents to be included in the modeling of human health risk. To compensate for some of the limitations in the waste data base, surrogate chemicals were selected to represent conservatively the most hazardous wastes potentially to be disposed of at the OWMC facility. This appendix describes in detail the procedure used to select the surrogate chemicals and estimate their concentrations in the organic waste streams to be managed by the facility. The chemicals used to model risks associated with emissions of metals and products of the incineration process, e.g. acid gases and products of incomplete combustion (PICs), such as dioxins and furans, were selected from empirical data compiled by the Ontario Research Foundation (ORF, 1986) and are not addressed here (see Appendix C for empirical data for metals, acid gases and dioxins/furans).

The starting point for the selection of surrogate organic constituents was the waste characterization data base developed by Canviro Associates (Canviro, 1985). The data base was developed from a survey of the types and quantities of special wastes generated by a sample of Ontario manufacturers. Waste in this survey has been categorized using the University of California at Davis (UCD) classification scheme, whereby waste streams are grouped in UCD

categories according to similarities in chemical constituents and physical state (see OWMC, 1982). The UCD categories are organized further into broader waste classes, which are listed in Table B-1. The surrogate selection process was used to identify the most hazardous constituent present in the waste streams of each UCD category of concern. In effect, each waste stream was represented by the most hazardous constituent, and each UCD category was represented by the most hazardous waste stream in that category. In this way, a surrogate chemical was selected to represent each UCD category in the modeling of environmental releases.

TABLE B-1

OWMC WASTE CLASSES

Class	Description
1	Organic sludges and still bottoms
2	Solvents and organic solutions
3	Oils and greases
4	Oil and water mixtures
5	Organic and oily residuals
6	Heavy metal solutions and residuals
7	Miscellaneous chemicals and products
8	Paint and organic residuals
9	Aqueous solutions and organics
10	Anion complexes
11	Sludges and inorganic residuals
12	Pesticide and herbicide waste
13	PCB wastes
14	Clean up residuals

Because this surrogate selection procedure was designed to identify organic constituents, the procedure was applied only to the UCD categories

which were considered to contain significant concentrations of organic chemicals. The UCD classes and categories included in the surrogate selection procedure (i.e., those waste streams considered to contain significant quanticies of organic constituents) are shown in Table B-2. In the case of Class 4, Oil and Water Mixtures, adequate data were not available from the Canviro data base to permit quantitative characterization of the surrogate chemicals. An alternative data base (Franklin Associates, 1983), which surveyed organic contaminants in waste oils, was used therefore to characterize this waste class. This data base was developed for the United States Environmental Protection Agency and was based on a relatively large number of samples of waste lubricants. Quantitative data were presented for concentrations in waste oils of a range of commonly used solvents, PCBs and

TABLE B-2

WASTE CATEGORIES INCLUDED IN THE ORGANIC CONSTITUENT SURROGATE SELECTION PROCEDURE

OWMC Class	UCD Category	Description
1	247	Halogenated still bottoms
	248	Still bottoms
	252	Solid or sludge tannery waste
	257	Phenolic wastes
	261	Polymeric resins
	263	Other polymeric wastes
	446	Degreasing sludge
	523	Tank bottom sediments
2	211	Halogenated solvents with heavy metals
	212	Halogenated solvents with other metals
	213	Other halogenated solvents
	214	Solvents without metal/non-metals
	216	Other non-halogenated solvents
	217	Unspecified solvents
	221	Organic liquid metals and halogens
	222	Organic liquids with halogens only
	223	Organic liquids with heavy metals
	224	Other organic liquids

TABLE B-2 (cont'd)

WASTE CATEGORIES INCLUDED IN THE ORGANIC CONSTITUENT SURROGATE SELECTION PROCEDURE

OWMC Class	UCD Category	Description
3	281	Waste oil and mixed oil
4	283	Oily mud, sediment and water
5	264	Thermoset wastes
	282	Oily tank bottoms
	284	Oily acidic solids
	285	Oily alkaline solids
	286	Oil/water separator sludge
	288	Oily wastes with heavy metals
	289	Oily wastes with water
	447	Tetraethyl lead sludge
7	145	Contaminated brine
	152	Inorganic solid chemicals
	243	Pharmaceutical wastes
	245	Off spec. or aged organics
	416	Ink sludge
	513	Lab. waste chemicals
8	254	Adhesives or glue
	258	Waste tar and residues
	262	Latex wastes
	454	Paint sludge
	455	Paint sludge with no heavy metals
9	225	Aqueous solutions, <10% organic residues
	226	Aqueous solutions, <10% halogenated residues
	227	Aqueous solutions, <10% organic residues
	228	Aqueous solutions, <10% halogenated residues
12	241	Pesticides and wastes
	532	Contaminated equipment and containers
13	242	PCB oil and high strength liquids
14	512	Spill cleanup
	521	Spent cartridge filters
	528	Pathological wastes
	531	Contaminated rags

other chemicals, and are summarized in Table B-3. While it was recognized that these data may not fully represent the organic contaminants to be found in Ontario wastes, in the absence of other sources of information they were considered a useful approximation.

TABLE B-3

CONCENTRATIONS OF ORGANIC CONTAMINANTS IN WASTE OILS

Contaminant	Total Samples Analyzed for Contaminant	Total Samples with Detectable Concentrations	Average of Values (mg/L)
PCBs	113	20	127
Benzo[a]pyrene	1	0	N/A
Naphthalene	1	1	440
1,1,1-Trichloroethane	95	7	1025
Trichloroethylene	101	90	527
Tetrachloroethylene	86	79	448
Dichlorodifluoro-methane	65	50	380
Benzene	1	1	160
Toluene	2	2	1260

Source: Franklin Associates (1983)

The waste characterization data described above were used in a series of separate ranking schemes developed for each source of environmental release modeled, i.e., fugitive emissions, truck spills, incinerator emissions, and tank farm fires. Each ranking scheme generated a hazard index to represent the degree of hazard associated with each chemical constituent of the waste

streams of concern. The constituent in each UCD category with the highest hazard index was selected as the surrogate for that UCD category. In general, the hazard rankings were based on three factors: (1) the toxicity of the constituent; (2) the potential for the constituent to be released to the environment; and (3) the quantity of chemical available for release. The hazard index for each constituent was calculated by multiplying a set of scores for the following three factors:

$$HI = R \times T \times Q$$

where HI = hazard index

R = release potential

T = toxicity

Q = quantity of chemical available for release

The hazard index resulting from the multiplication of these scores was thus designed to provide a systematic means for comparing the relative hazard presented by waste constituents.

B.2 BASIS FOR HAZARD INDEX

To represent each of the general factors comprising the hazard indices described above, chemical specific parameters were chosen. The specific parameters representing toxicity and release potential were different, depending upon the source of release under consideration. The basis for the hazard indices derived for each source of environmental release and the

specific parameters used to derive the hazard indices are summarized in Table B-4. Operationally, a scoring scheme was developed for each parameter used in the calculation of the hazard indices. The scores for the appropriate paramecers representing toxicity, release potential and quantity were then multiplied resulting in the hazard index. The following sections describe the basis for the parameters and scoring systems used to calculate the hazard indices.

TABLE B-4

BASIS FOR HAZARD INDICES

Source of Release	Hazard Index Basis	Parameter
Fugitive Emissions	Release Potential	Vapor Pressure
	Toxicity	Chronic Toxicity
	Quantity	Chemical Quantity
Incinerator Emissions	Toxicity	Chronic Toxicity
	Quantity	Chemical Quantity
Truck Spill	Release Potential	Vapor Pressure
	Toxicity	Acute Toxicity
	Quantity	Chemical Quantity
Tank Farm Fire	Release Potential	Heat of Vaporization
	Toxicity	Acute Toxicity
	Quantity	Chemical Quantity

RELEASE POTENTIAL

Vapor Pressure

The parameter used to represent release potential for the truck spill and fugitive emission releases was selected by examining the equations used to calculate emission rates. For example, the equation used to calculate the emission rates for truck spills was (see Appendix D, Volume 2):

$$Q_i = \frac{(Kg_i)(A)(P_i)(M_i)(x_i)}{(R)(T)}$$

where Q = emission rate for species, g/s

Kg_i = the gas mass transfer coefficient, m/s

A = area of spill, m^2

P_i = vapor pressure of the chemical, Pa

M_i = molecular weight of chemical, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

x_i = mole fraction of species i in waste

The chemical-specific parameters in the above equation are gas mass transfer coefficient, vapor pressure, molecular weight and mole fraction. Similarly, for the fugitive emissions, the emission rates are typically a function of vapor pressure, molecular weight and mole fraction. For example, the losses from storage tanks are given by (see Appendix C):

$$Q_i = (4.91 \times 10^{-6})(P_i)(M_i)(x_i)$$

where: Q = emission rate for species i, g/s

P_i = vapor pressure of chemical, Pa

M_i = molecular weight of chemical, g/mol

x_i = mole fraction of species i in waste

Mole fraction appears in both equations and is treated separately under the heading of "quantity". Of the other parameters, vapor pressure was considered to be the primary parameter differentiating between emission rates for various chemicals. Vapor pressure scores then were assigned on a scale of 1 to 10 as shown in Table B-5.

TABLE B-5

VAPOR PRESSURE SCORES

Vapor Pressure (mm Hg)	Score
>100	10
50-100	9
10-50	8
5-10	7
1-5	6
0.5-1	5
0.1-0.5	4
0.05-0.01	3
0.01-0.05	2
<.01	1

In the release of chemicals via incinerator emissions, all organic wastes were assumed to be in the vapor phase at the high temperatures involved in the incinerator process. For this reason, release potential was not included in the calculation of the hazard index for the incinerator emissions source of release.

Heat of Vaporization

Similar to the truck spill and fugitive emission releases, the parameter representing release potential for tank farm fire releases was selected by review of the emission rate equation (see Section 3.4, Volume 1):

$$Q_i = \frac{(21\ 000)\ (F)\ (Aw)^{0.82}}{\Delta H_i}$$

where Q = emission rate for species i, lb/h

F = insulation factor, 1.0 for a base vessel

Aw = wetted surface area of tank, m^2

ΔH_i = heat of vaporization for species i, BTU/lb

Since the heat of vaporization is the only chemical-specific parameter in this equation, the reciprocal of heat of vaporization ($1/\Delta H$) was used to represent release potential in the calculation of the Hazard Index.

TOXICITY

Acute Toxicity

Acute toxicity was chosen to represent toxicity for the truck spill and tank farm fire releases. In both of these scenarios, a large quantity of chemical is released over a relatively short period of time. Thus, acute effects of the chemicals would be of primary concern.

The acute toxicity score was based on LD_{50} values. The LD_{50} represents that dose which is, on average, lethal to fifty percent of the test population following a single exposure (Doull et al., 1980). For consistency, the lowest LD_{50} reported for a mammalian species was used in the ranking.

The LD_{50} values were assigned scores according to the classes defined by Gosselin, Smith and Hodge (1984) as shown in Table B-6.

TABLE B-6

ACUTE TOXICITY SCORES

Score	Description	LD_{50}(mg/kg)
1	Practically non toxic	>15000
2	Slightly toxic	500-15000
3	Moderately toxic	500-5000
4	Very toxic	50-500
5	Extremely toxic	5-50
6	Super toxic	<5

Chronic Toxicity

For releases via fugitive and incinerator emissions, chronic toxicity was chosen to represent toxicity. In both of these scenarios, relatively small emissions of chemicals occur over a long period of time. Thus, chronic effects of the chemicals would be of primary concern.

The chemicals were ranked according to their chronic toxicity using Acceptable Daily Intakes (ADIs) for noncarcinogens and Unit Cancer Risks (UCRs) for carcinogens as derived in Appendix A. Because these measures of risk are expressed using different parameters, it was necessary to develop a method for scoring both carcinogenic and noncarcinogenic compounds using the same scale. This was accomplished by converting the UCRs to a Risk Specific Dose (RSD) using the following approach.

By assuming that the dose-response curve for carcinogens is a straight line at very low doses, an RSD can be calculated for carcinogens using the formula:

$$RSD = \frac{\text{Level of Risk}}{UCR}$$

To derive an RSD from a UCR, a set of assumptions can then be made regarding the level of risk considered to be acceptable, and the level of risk associated with noncarcinogenic ADIs. Using these assumptions, a level of risk can be chosen which will enable the direct comparison of carcinogenic and noncarcinogenic toxic potential.

Carcinogens, under the assumptions used to derive UCRs, present a non-zero risk at all exposure levels above zero. Nevertheless, it is commonly accepted in regulatory decision-making that for all carcinogens there is an exposure range under which the magnitude of the risk is very small. In many cases, lifetime risks falling below the range of 10^{-5} to 10^{-6} have been considered very small. These probabilities reflect highly unlikely events, especially when it is further considered that they are predicted using extrapolation methods designed to produce upper bound estimates.

ADIs are thought to reflect conditions of exposure to noncarcinogens that are without risk. It is not possible, however, to claim that an ADI carries absolutely no risk for the human population. At best, all that can be claimed for an ADI is that any associated risk is likely to be very small. No means are available to quantify this "very small" risk. In the absence of evidence to the contrary and to provide a systematic means for comparing carcinogenic and noncarcinogenic compounds (so as to avoid having to create

two different toxicity scales), it was assumed that the "risk" associated with an ADI is in the same range (10^{-5} to 10^{-6}) as that considered to be "very small" for carcinogens. This assumption should not be used outside the context of this specific scoring objective. Neither should it be taken to mean that the actual risk to a target population at an ADI exposure level is in the range assumed here. This assumption has been used only to provide a systematic means of aligning and comparing the risks associated with carcinogenic and noncarcinogenic compounds for this study.

It is further assumed that exposure at an RSD for a noncarcinogen will ensure that there is no more than a 10^{-6} (1 in 1 000 000) risk of a toxic effect occurring. An RSD derived for carcinogens using a risk level of 10^{-6} will represent the dose that will give rise to the same maximum lifetime risk (10^{-6}). Carcinogens and noncarcinogens can then be directly compared because they will have been adjusted for potency and represent the same risk level. Using this methodology, scores were assigned according to order of magnitude changes in the RSDs or ADIs as shown in Table B-7.

TABLE B-7

CHRONIC TOXICITY SCORES

RSD or ADI (mg/kg/day)			Score
		$\leq 10^{-7}$	9
$>10^{-7}$	-	$\leq 10^{-6}$	8
$>10^{-6}$	-	$\leq 10^{-5}$	7
$>10^{-5}$	-	$\leq 10^{-4}$	6
$>10^{-4}$	-	$\leq 10^{-3}$	5
$>10^{-3}$	-	$\leq 10^{-2}$	4
$>10^{-2}$	-	$\leq 10^{-1}$	3
$>10^{-1}$	-	≤ 1	2
>1			1

QUANTITY

For all sources of release, the quantity of the constituent was used to represent quantity. The quantity of each constituent was derived from the waste stream data in the Canviro database with the exception of Class 4 wastes which were characterized using the data from Franklin Associates. The objective of this procedure was to estimate annual volumes of specific constituents of waste streams associated with each UCD category. Aggregation of constituent quantities was restricted to wastes within UCD categories because only within these categories were wastes considered generically similar. Thus, the following three-step procedure was applied to each of the UCD categories listed in Table B-2.

Step (1) Calculate quantity of each individual waste stream. Because of a concern that quantities of individual waste streams projected in the waste profile might underestimate the actual quantities, the approach adopted was to give equal status to each waste stream. Individual waste stream quantity was thus approximated as the extrapolated quantity of the pertinent UCD category in the waste profile divided by the number of wastes identified in the data base for that category.

Step (2) Calculate constituent quantity for each waste stream. Constituent quantity was calculated by multiplying the constituent concentration reported in the Canviro data base by the waste stream quantity estimated in Step 1.

Step (3) Generate aggregated constituent quantity for each UCD category. Quantities of each constituent were summed for all waste streams within each UCD category generating an aggregated quantity of each hazardous constituent present in the UCD category.

The aggregated quantities obtained were then placed on a scale of 1-6 as shown in Table B-8. In this manner, a numerical quantity score was obtained for those chemicals appearing as waste constituents in the Canviro data base.

TABLE B-8

QUANTITY SCORES

Quantity (t/a)	Score
>500	6
101-500	5
51-100	4
11-50	3
1-10	2
<1	1

B.3 RESULTS OF SURROGATE SELECTION

SELECTION OF SURROGATE CHEMICALS

The results of the surrogate selection process are summarized in a series of tables:

(1) The parameters and associated scores used to select surrogate chemicals are shown in Table B-9.

(2) The basis for the chronic toxicity values (ADIs or RSDs) are summarized in Table B-10.

(3) The chemicals scoring the highest hazard index within each UCD class are shown in Table B-11. We note that in the case of the chronic emissions associated with fugitive releases and incineration, all UCD categories were assumed to contribute to the overall emissions. The surrogate for each UCD category, therefore, was selected for use in the subsequent modeling of emissions.

TABLE B-9

ENVIRONMENTAL PARAMETERS AND ASSOCIATED SCORES*

Compound	RSD or ADI (mg/kg/day)	Score	LD50 (mg/day)	Score	Vapor Pressure (mm Hg)	V.P. Temp. (C)	Score	Heat of Vaporization (kJ/Mole)	Score
Acetone	2.86E+00	1	9750 (7)	2	2.40E+02 (1)	25	10	31.3 (1)	0.032
Acrylonitrile	4.17E-06	7	27 (7)	5	1.03E+02 (1)	25	10	32.6 (1)	0.031
Benzene	1.92E-05	6	3800 (7)	3	9.39E+01 (1)	25	9	30.7 (1)	0.033
Chlorobenzene	1.40E-02	3	2830 (7)	3	1.19E+01 (1)	25	8	36.5 (1)	0.027
Chloroform	1.33E-05	6	800 (7)	3	1.96E+02 (1)	25	10	29.7 (1)	0.034
Cresols	1.13E-01	2	242 (7)	4	1.43E-01 (1)	25	4	46.7 (1)	0.021
o-Dichlorobenzene	9.00E-02	3	414 (7)	4	1.39E+00 (1)	25	6	39.7 (1)	0.025
Dichlorodifluoromethane	8.00E-01	2	47500 (7)	1	4.87E+03 (3)	25	10	24.4 (1)	0.041
Ethanol	4.95E-02	3	5560 (7)	2	5.88E+01 (1)	25	9	38.7 (1)	0.026
Ethylene glycol	1.00E+00	1	7500 (7)	2	5.02E-02 (2)	20	3	52.5 (1)	0.019
Fluorocarbon 113	5.02E+01	1	43000 (7)	1	3.32E+02 (1)	25	10	27.5 (1)	0.036
Formaldehyde	8.93E-05	6	260 (7)	4	3.83E+03 (1)	25	10	23 (1)	0.043
Glycerol	1.70E-02	3	7750 (7)	2	1.37E-05 (1)	25	1	61.1 (1)	0.016
Hexachlorobenzene	5.99E-07	8	10000 (7)	2	1.10E-05 (3)	20	1	64 (6)	0.016
n-Hexane	5.20E+00	1	28710 (7)	1	1.51E+02 (1)	25	10	28.8 (1)	0.035
Hydrazine	2.56E-07	8	91 (7)	4	1.42E+01 (1)	25	8	44.8 (1)	0.022
Isopropyl alcohol	6.40E-02	3	5840 (7)	2	4.52E+01 (1)	25	8	39.8 (1)	0.025
Kerosene	2.40E-02	3	20000 (7)	1	5.00E+00 (5)	25	7	24 (6)	0.042
Methyl ethyl ketone	2.00E-02	3	2740 (7)	3	9.41E+01 (1)	25	9	31.2 (1)	0.032
Methyl isobutyl ketone	1.04E-01	2	2080 (7)	3	1.96E+01 (1)	25	8	35.5 (1)	0.028
Methylene chloride	1.59E-03	4	167 (7)	4	4.36E+02 (1)	25	10	27.9 (1)	0.036
Napthalene	2.57E-01	2	1780 (7)	3	8.70E-02 (3)	25	3	43.3 (1)	0.023
PCB	2.30E-07	8	1010 (7)	3	2.42E-04 (4)	25	1	54 (6)	0.019
Petroleum distillates	1.12E-01	2	1240 (7)	3	4.00E+00 (5)	25	6	31 (6)	0.032
Phenol	1.10E-01	2	300 (7)	4	5.00E-01 (2)	25	5	45.6 (1)	0.022
Pyridine	9.00E-03	4	891 (7)	3	2.03E+01 (1)	25	8	35.1 (1)	0.028
Styrene	9.50E-01	2	316 (7)	4	5.89E+00 (1)	25	7	36.8 (1)	0.027
Tetrachloroethylene	2.86E-05	6	8100 (7)	2	1.89E+01 (1)	25	8	34.7 (1)	0.029
Tetraethyl lead	2.80E-07	8	15 (7)	5	1.50E-01 (2)	20	4	54 (8)	0.019
Toluene	4.28E-01	2	5000 (7)	3	2.74E+01 (1)	25	8	33.1 (1)	0.030
1,1,1-Trichloroethane	6.25E-04	5	750 (7)	3	2.40E+01 (1)	25	8	33.3 (1)	0.030
Trichloroethylene	5.26E-05	6	4920 (7)	3	7.45E+01 (1)	25	9	31.3 (1)	0.032
Xylene	2.29E+00	1	4300 (7)	3	7.79E+00 (1)	25	7	36.4 (1)	0.027

* References are in parentheses:

(1) Reid et al., 1977
(2) Verschueren, 1983
(3) Mabey et al., 1982
(4) NAS, 1979
(5) GE Manual, 1980
(6) Calculated
(7) NIOSH, 1983
(8) Weast, 1984
(9) CRC, 1984

TABLE B-10

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD or ADI (mg/kg/day)	SCORE	BASIS	REFERENCE
Acetone	2.86	1	USEPA ADI based on TLV associated with minor irritation.	USEPA, 1984
Acrylonitrile	4.17×10^{-6}	7	USEPA unit risk value based on human occupational exposure.	CAG, 1984
Benzene	1.92×10^{-5}	6	USEPA unit risk based on human occupational exposure.	CAG, 1984
Chlorobenzene	1.40×10^{-2}	3	USEPA ADI based on rat subchronic NOEL; higher doses caused histopathological changes in the liver.	USEPA, 1984
Chloroform	1.33×10^{-5}	6	USEPA unit risk value based on oral animal study	CAG, 1984
Cresols	0.113	2	USEPA ADI based on TLV	USEPA, 1984
O-Dichlorobenzene	9.0×10^{-2}	3	USEPA ADI based on rat subchronic oral NOEL higher doses caused increased liver and kidney weights and hepatic necrosis	USEPA, 1984
Ethanol	4.95×10^{-2}	3	Rat 90 day inhalation NOEL; higher doses induced cirrhois of the liver	See Appendix A
Ethylene glycol	1.0	1	Rat 2 year oral NOEL; higher doses produced calcification of the kidneys and renal injury	Morris et al as cited in Clayton and Clayton 1981-
Fluorocarbon 113	50.2	1	Rabbit, 12-24 month NOEL; higher doses produced fatty infiltration of the liver	Reinke 1962 as cited in ACGIH 1981

TABLE B-10 (cont'd)

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD OR ADI (mg/kg/day)	SCORE	BASIS	Reference
Formaldehyde	8.93×10^{-5}	6	USEPA unit risk based on nasal carcinomas	See Appendix
Glycerol	1.7×10^{-2}	3	TLV	ACGIH 1981
Hexachlorobenzene	5.99×10^{-7}	8	USEPA unit risk based on oral animal study	CAG, 1984
n-Hexane	5.2	1	Mouse, 1 yr NOEL; higher doses produced neurotoxic effects	Miyagaki as cited in Spencer et al. 1980
Hydrazine	2.56×10^{-7}	8	USEPA VCR value based on occurence of adenocarcinomas o the lung	USEPA, 1982
Isopropyl alcohol	6.4×10^{-2}	3	Human 6 week oral NOEL; higher doses in a different study caused CNS effects such as ataxia prostration and narcosis	See Appendix A
Kerosene	2.4×10^{-2}	3	NIOSH recommended standard	NIOSH 1977 as cited in Clayton and Clayton 1981-1982
Methyl ethyl ketone	2.00×10^{-2}	3	USEPA ADI based on subchronic rat inhalation NOEL; higher doses caused teratogenic effects in animals	USEPA, 1984
Methyl isobutyl ketone	0.104	2	USEPA ADI based on TLV associated with liver and kidney damage in rats	USEPA, 1984
Methylene chloride	1.59×10^{-3}	4	USEPA unit risk based on oral animal study	CAG, 1984
Naphthalene	0.257	2	USEPA ADI based on TLV; higher doses caused headache, eye injury and kidney damage	USEPA, 1984

TABLE B-10 (cont'd)

BASIS FOR CHRONIC TOXICITY SCORES

COMPOUND	RSD OR ADI (mg/kg/day)	SCORE	BASIS	Reference
PCB-1254	2.3×10^{-7}	8	USEPA unit risk based on animal study	CAG, 1984
Phenol	0.11	2	USEPA ADI based on rat subchronic to chronic oral LOEL associated with slight kidney damage	USEPA, 1984
Petroleum Distillates	1.12×10^{-1}	2	Monkey, 90 day NOEL associated with a decline in body weight	See Appendix A
Pyridine	9.0×10^{-3}	4	Rat 6 month inhalation NOEL associated with increased liver weight	See Appendix A
Styrene	0.95	2	Rat 185 day oral NOEL; higher doses caused liver and kidney weight deviations	See Appendix A
Tetrachloroe-ethylene	2.86×10^{-5}	6	USEPA unit risk value based on oral animal study	CAG, 1984
Tetraethyl lead	2.8×10^{-7}	8	Rat 20 week oral LOEL associated with histological changes in liver, pancreas, renal, endocrine and nervous system	See Appendix A
Toluene	0.428	2	USEPA ADI value based on rat chronic oral NOEL; higher doses have not been tested	USEPA, 1984
Trichloroethylene	5.26×10^{-5}	6	USEPA unit risk based on oral animal study	CAG, 1984
1,1,1-Trichloro-ethane	6.25×10^{-4}	5	USEPA unit risk based on oral animal study	CAG, 1984
Xylene	2.29	1	USEPA ADI based on TLV; higher inhaled doses caused maternal toxicity in a rat teratology study	USEPA, 1984

TABLE B-11

CHEMICALS SCORING THE HIGHEST HAZARD INDEX

UCD Category	Fugitive Emissions	Score	Incinerator Emissions	Score	Truck Spills	Score	Tank Farm Fire	Score
211	Trichloroethylene	270	Tetrachloroethylene Trichloroethylene	30 30	Methylene chloride	200	Methylene chloride	0.717
212	Trichloroethylene	162	Trichloroethylene Tetrachloroethylene	18 18	Trichloroethylene	81	Trichloroethylene	0.288
213	Chloroform	240	1,1,1-Trichloroethane	25	Methylene chloride	200	Methylene chloride	0.717
214	Petroleum distillates	72	Petroleum distillates	12	Petroleum distillates	108	Petroleum distillates	0.581
215	Toluene	80	Toluene	10	Toluene	120	Toluene	0.453
216	Benzene	108	Benzene	12	Toluene	96	Petroleum distillates	0.484
217	Ethanol	135	Ethanol	15	Ethanol	90	Ethanol	0.258
224	Acrylonitrile	140	Hydrazine	16	Acrylonitrile	100	Acrylonitrile	0.307
225	Formaldehyde	120	Formaldehyde	12	Formaldehyde	80	Formaldehyde	0.348
227	Methyl ethyl ketone	54	Toluene Methyl ethyl ketone	6 6	Toluene	72	Toluene	0.272
241	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
242	PCB	32	PCB	32	PCB	12	PCB	0.222
243	Ethanol	81	Ethanol	9	Ethanol Petroleum distillates	54 54	Petroleum distillates	0.290
245	Formaldehyde	120	Ethanol Formaldehyde	12 12	Formaldehyde	80	Formaldehyde	0.348
247	Methylene chloride	80	Methylene chloride	8	Methylene chloride	80	Methylene chloride	0.287
248	Ethanol	135	Ethanol	15	Petroleum distillates	108	Petroleum distillates	0.581
254	1,1,1-Trichloroethane Methylene chloride	120 120	1,1,1-Trichloroethane	15	Methylene chloride	120	Methylene chloride	0.430
257	Formaldehyde	120	Phenol Formaldehyde	12 12	Phenol	120	Phenol	0.526
261	Styrene	56	Styrene Cresols	8 8	Styrene	112	Styrene	0.435
262	Formaldehyde	60	Formaldehyde	6	Formaldehyde	40	Formaldehyde	0.174
263	Acrylonitrile	140	Acrylonitrile	14	Acrylonitrile	100	Acrylonitrile	0.307
264	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290

TABLE B-11 (cont'd)

CHEMICALS SCORING THE HIGHEST HAZARD INDEX

UCD Category	Fugitive Emissions	Score	Incinerator Emissions	Score	Truck Spills	Score	Tank Farm Fire	Score
281	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
283	Trichloroethylene	108	PCB	16	Trichloroethylene	54	Benzene	0.195
	Benzene	108			Benzene	54		
286	Phenol	20	Phenol	4	Phenol	40	Phenol	0.175
289	Petroleum distillates	36	Petroleum distillates	6	Petroleum distillates	54	Petroleum distillates	0.290
416	Ethanol	81	Ethanol	9	Ethanol	54	Petroleum distillates	0.194
446	Trichloroethylene	270	Tetrachloroethylene	30	Trichloroethylene	135	Trichloroethylene	0.479
			Trichloroethylene	30				
447	Tetraethyl lead	32	Tetraethyl lead	8	Tetraethyl lead	20	Tetraethyl lead	0.093
454	Petroleum distillates	72	Petroleum distillates	12	Petroleum distillates	108	Petroleum distillates	0.581
455	Petroleum distillates	48	Petroleum distillates	8	Petroleum distillates	72	Petroleum distillates	0.387
513	Pyridine	96	Pyridine	12	Pyridine	72	Petroleum distillates	0.290
521	Toluene	16	Toluene	2	Toluene	24	Petroleum distillates	0.097
523	Formaldehyde	120	Formaldehyde	12	Formaldehyde	80	Formaldehyde	0.348
531	Petroleum distillates	60	Petroleum distillates	10	Petroleum distillates	90	Petroleum distillates	0.484

In the case of episodic releases associated with truck spills and the tank farm fire, the situation is different, since any given event will only involve a single UCD category, i.e., that category being transported or stored. Consequently, for the episodic releases, only the constituent with the highest hazard index (methylene chloride as shown in Table B-11) was selected for subsequent modeling.

CALCULATION OF MASS FRACTIONS

The final step in the surrogate selection process was the generation of mass fractions to be used in the calculation of air emissions (see Appendix C for emission equations). Again, different mass fractions were generated for each source of environmental release. For the chronic emissions, i.e., incinerator and fugitive emissions, it was assumed for the purposes of modeling that all of the waste streams are processed together essentially as a mixture. The mass fraction, therefore, was calculated by summing all of the estimated quantities for each surrogate chemical and dividing this sum by the total quantity of waste processed by the facility. In the case of a "tie" (two or more chemicals producing the same hazard index), the quantity of each surrogate was adjusted accordingly. For example, if two chemicals tied for a given UCD category, the quantity associated with the UCD category was split equally between the two chemicals. The mass fractions for fugitive and incinerator emissions are shown in Table B-12.

For the truck spill scenario, it was assumed that each waste stream would be transported separately. Therefore, the mass fraction was calculated simply as the average concentration of the surrogate chemical in that UCD category. Similarly, for the tank farm fire, it was assumed that each waste stream could

TABLE B-12

MASS FRACTIONS FOR FUGITIVE AND INCINERATOR EMISSIONS

Fugitive Emissions		Incinerator Emissions	
Compound	Mass Fraction	Compound	Mass Fraction
Acrylonitrile	1.01E-03	Acrylonitrile	3.37E-04
Benzene	2.03E-03	Benzene	2.02E-03
Chloroform	8.42E-03	Cresols	1.51E-02
Ethanol	5.88E-02	Ethanol	5.56E-02
Formaldehyde	1.04E-03	Formaldehyde	1.30E-03
Methyl ethyl ketone	3.37E-04	Hydrazine	3.37E-04
Methylene chloride	3.47E-03	Methyl ethyl ketone	1.68E-04
PCB	1.06E-03	Methylene chloride	1.01E-04
Petroleum distillates	6.04E-02	PCB	1.08E-03
Phenol	5.05E-05	Petroleum distillates	6.04E-02
Pyridine	2.83E-03	Phenol	4.93E-03
Styrene	2.83E-02	Pyridine	2.83E-03
Tetraethyl lead	1.68E-06	Styrene	1.41E-02
Toluene	8.00E-03	Tetrachloroethylene	1.03E-02
1,1,1-Trichloroethane	3.37E-03	Tetraethyl lead	1.68E-06
Trichloroethylene	1.98E-02	Toluene	9.67E-03
		1,1,1-Trichloroethane	1.82E-02
		Trichloroethylene	9.90E-03

be the only waste stream present in a holding tank. Thus, the mass fraction was calculated as the average concentration of the surrogate chemical in each UCD category. The mass fractions for the tank farm fire and truck spills are shown by UCD category in Table B-13.

B.4 LIMITATIONS OF SURROGATE SELECTION PROCEDURE

In applying the ranking scheme, the following limitations of the data were noted:

(1) The data base was developed from a partial survey of Ontario industries and may not be fully representative of the types of hazardous wastes generated in Ontario. In some cases, the sample of waste addressed within a UCD category by Canviro was only a small fraction of the total waste projection for that UCD category, making any direct extrapolation of quantities or chemical types questionable. For example, UCD category 247 includes four waste streams totalling 9.32 tonnes per annum compared with a total of 600 tonnes per annum projected for the facility.

(2) The amount of quantitative waste composition data suitable for use in a ranking scheme was limited. This limitation arises in two ways. In some cases, hazardous waste constituents were identified, but without quantitative concentrations. In other cases, where quantitative concentrations were provided, the constituents were described only in generic terms (e.g., aromatic hydrocarbons) as opposed to a specific chemical compound. Where feasible, this latter problem was addressed by making reasonable assumptions about

TABLE B-13

MASS FRACTIONS FOR TRUCK SPILLS AND TANK FARM FIRES

Truck Spill			Tank Farm Fire		
SCD Category	Compound	Mass Fraction	SCD Category	Compound	Mass Fraction
211	Methylene chloride	0.62	211	Methylene chloride	0.62
212	Trichloroethylene	0.49	212	Trichloroethylene	0.49
213	Methylene chloride	0.68	213	Methylene chloride	0.68
214	Petroleum distillates	0.63	214	Petroleum distillates	0.63
215	Toluene	0.79	215	Toluene	0.79
216	Toluene	0.4	216	Petroleum distillates	0.51
217	Ethanol	0.58	217	Ethanol	0.58
224	Acrylonitrile	0.1	224	Acrylonitrile	0.1
225	Formaldehyde	0.01	225	Formaldehyde	0.01
227	Toluene	0.2	227	Toluene	0.2
241	Petroleum distillates	0.8	241	Petroleum distillates	0.8
242	PCB	0.79	242	PCB	0.79
243	Ethanol	0.18	243	Petroleum distillates	0.5
243	Petroleum distillates	0.5	245	Formaldehyde	0.04
245	Formaldehyde	0.04	247	Methylene chloride	0.01
247	Methylene chloride	0.01	248	Petroleum distillates	0.55
248	Petroleum distillates	0.55	254	Methylene chloride	0.4
254	Methylene chloride	0.4	257	Phenol	0.29
257	Phenol	0.29	261	Styrene	0.28
261	Styrene	0.28	262	Formaldehyde	0.02
262	Formaldehyde	0.02	263	Acrylonitrile	0.005
263	Acrylonitrile	0.005	264	Petroleum distillates	0.56
264	Petroleum distillates	0.56	281	Petroleum distillates	0.32
281	Petroleum distillates	0.32	283	Benzene	0.00016
283	Trichloroethylene	0.000527	286	Phenol	0.005
283	Benzene	0.00016	289	Petroleum distillates	0.07
286	Phenol	0.005	416	Petroleum distillates	0.1
289	Petroleum distillates	0.07	446	Trichloroethylene	0.28
416	Ethanol	0.45	447	Tetraethyl lead	0.01
446	Trichloroethylene	0.28	454	Petroleum distillates	0.22
447	Tetraethyl lead	0.01	455	Petroleum distillates	0.18
454	Petroleum distillates	0.22	513	Petroleum distillates	0.99
455	Petroleum distillates	0.18	521	Petroleum distillates	0.001
513	Pyridine	0.42	523	Formaldehyde	0.001
521	Toluene	0.001	531	Petroleum distillates	0.33
523	Formaldehyde	0.001			
531	Petroleum distillates	0.33			

the meaning of the generic terms, e.g., "phenols", were assumed to be phenol and "chlorinated ethylenes" were assumed to be a 50/50 mixture of trichloroethylene and tetrachloroethylene. While this was feasible in some instances, some categories of wastes could only be characterized in a very general way, e.g. herbicides and pesticides.

The net effect of this lack of specificity was to limit the number of waste streams that could be quantitatively characterized to approximately 60% of the total number (390) containing organic chemicals addressed by the Canviro survey. The amount of usable data contained in the Canviro data base is summarized by waste stream class in Table B-14.

The total number of data points (each corresponding to a constituent) in the Canviro data base is shown in the second column. The third column lists the number of those data points for which an associated concentration was cited. Finally, the number of data points that were ultimately used is listed in the fourth column. In cases where hazardous waste constituents were identified without quantitative concentrations, waste streams with concentrations in the same category were used to extrapolate to the deficient streams. In one case (class 13), the extrapolation procedure resulted in the number of usable data points exceeding the number of data points with available concentrations. As can be seen, 425 of 2659 (or about 16%) of the data points listed in the data base were usable for the purposes of this assessment.

TABLE B-14

SUMMARY OF USABLE DATA POINTS FROM THE CANVIRO DATA BASE

Waste Stream Class	Total Data Points	Data Points With Conc.	Usable Data Points
1	320	185	51
2	668	421	242
3	403	343	7
4	DATA TAKEN FROM FRANKLIN ASSOCIATES (1983)		
5	313	260	9
7	270	131	33
8	347	207	42
9	33	30	7
12	17	12	1
13	27	8	19
14	261	102	14
TOTAL	2659	1699	425

REFERENCES

American Conference of Government Industrial Hygienists (ACGIH). 1981. Documentation of the Threshold Limit Values. 4th ed. Supplemental Documentation, 1983. ACGIH, Cincinnati, Ohio.

Canviro Consultants, Ltd. 1985. Market Assessment Program: Survey of 1000 Industrial Establishments. Prepared for Ontario Waste Management Corporation.

Carcinogen Assessment Group (CAG). 1984. Relative Carcinogenic Potencies Among 53 Chemicals Evaluated by the Carcinogen Assessment Group as Suspect Carcinogens. From Draft EPA Health Assessment Document for Carbon Tetrachloride. September.

Clayton, G.D. and Clayton, F.E. 1981-1982. Patty's Industrial Hygiene and Toxicology. Third Revised Edition. Vols. 2A, 2B, and 2C: Toxicology. John Wiley and Sons, New York.

CRC. 1984. Handbook of Chemistry and Physics, 64th Edition. Ed. Robert C. Weast, Ph.D.

Doull et al. 1980. Casarett and Doull's Toxicology. Second Edition. MacMillan Publishing Co., Inc., New York.

Franklin Associates, Ltd. 1983. Revised Draft Report on Waste Oil Management and Composition. USEPA Office of Solid Waste.

General Electric Co. (GE). 1980. Material Safety Data Sheets. Nielsen, J.M. ed.

Gosselin, Smith, & Hodge. 1984. Clinical Toxicology of Commercial Products. Fifth Edition. Williams and Wilkins, Baltimore.

Mabey, W.R., Smith, J.H., Podoll, R.T., Johnson, H.L., Mill, T., Chou, T.W., Gates, J., Partridge, I.W., Jaber, H., and Vandenberg, O. 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, D.C. EPA Contracts 68-01-3867 and 68-03-2981.

National Academy of Sciences (NAS). 1979. Polychlorinated Biphenyls. Environmental Studies Board Commission on Natural Resources, National Research Council. Washington, D.C.

National Institute for Occupational Safety and Health (NIOSH). 1983. Registry of Toxic Effects of Chemical Substances (RTECS), 1981-1982 ed. Vols. 1-3. DHHS (NIOSH) Publication No. 33-107

Ontario Research Foundation. 1985. Emissions Related Studies: Preliminary Estimates of Air Emissions. In preparation for Ontario Waste Management Corporation.

Ontario Waste Management Corporation. 1982. Facilities Development Process. Phase 1 Report. September.

Reid, R.C., Prausnitz, J.M. and Sherwood, T.K. 1977. Properties of Gases and Liquids. McGraw-Hill Book Co., New York

Spencer, P.S., Schaumburg, H.S., Sabri, M.I. and Veronesi, B. 1980. The enlarging view of hexacarbon neurotoxicity. CRC Crit. Rev. in Tox. 7:279-357

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/ February. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

United States Environmental Protection Agency. 1982. Health Effects Assessment Summary for 300 Hazardous Organic Constituents. In support of Regulatory Impact Analysis of the Land Disposal Branch and Interim Final Incinerator Regulations of the Technical Branch of the Office of Solid Waste. Draft, September.

USEPA, 1984. Summary of Current Acceptable Daily Intakes (ADIs) of Oral Exposure. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. 2nd ed. Van Nostrand Reinhold Co., New York.

APPENDIX C

ESTIMATION OF ENVIRONMENTAL RELEASES

C O N T E N T S

C.1	INTRODUCTION	
	C.1.1 Emission Sources	C-1
	C.1.2 Assumptions for Calculations	C-2
C.2	WASTE TRANSPORTATION	C-8
C.3	BULK WASTE UNLOADING AND TRANSFER	C-11
	C.3.1 Organic Waste Reception - Normal Operation	C-13
	C.3.2 Organic Waste Reception - Failure	C-15
	C.3.3 Inorganic Waste Reception - Normal Operation	C-15
	C.3.4 Inorganic Waste Reception - Failure	C-16
	C.3.5 Routine Spills	C-17
C.4	DRUM UNLOADING AND TRANSFER	C-20
	C.4.1 Drum Opening	C-21
	C.4.2 Drum Transfer	C-23
C.5	WASTE STORAGE-TANKS	C-27
	C.5.1 Organic Waste Storage	C-28
	C.5.2 Inorganic Waste Storage	C-30
	C.5.3 Tank Farm Fire	C-31
C.6	PHYSICAL CHEMICAL TREATMENT	C-32
	C.6.1 Dissolved Air Flotation	C-33
	C.6.2 Emulsion Breaking	C-35
	C.6.3 Cyanide Treatment	C-36
	C.6.4 Sulfide Treatment	C-38
	C.6.5 Mixing of Incompatible Wastes	C-40
C.7	INCINERATION	C-40
	C.7.1 Controlled Stack Emissions	C-41
	C.7.2 Incinerator Scrubber Failure	C-45
C.8	SUMMARY OF RESULTS	C-46
	REFERENCES	C-52

TABLES

		Page
C-1	Mole Fractions for Estimation of Emission Rates	C-5
C-2	Chemical Property Data for Estimation of Emission Rates	C-7
C-3	Stack Emission Estimates - Metals	C-43
C-4	Stack Emission Estimates - Acid Gas	C-44
C-5	Stack Emission Estimates - PICs	C-45
C-6	Stack Emission Estimates - Scrubber Failure	C-46
C-7	Fugitive Emission Rates at 150 000 t/a	C-47
C-8	Fugitive Emission Rates at 300 000 t/a	C-48
C-9	Incinerator Stack Emission Estimates	C-49
C-10	Emission Estimates for Upset Scenarios	C-50

C.1 INTRODUCTION

C.1.1 EMISSION SOURCES

The proposed OWMC facility will consist of four major components: an incinerator, a physical-chemical treatment plant, a solidification plant and an engineered landfill, along with the associated transportation, reception and storage facilities (See Monenco, 1985). Air emissions will be generated from these sources under four possible scenarios: normal operating conditions, facility upset conditions, on-site accidents and off-site transportation accidents. The focus of the emission estimates was primarily in gaseous pollutants (in particular volatile organic chemicals) released under these scenarios, although consideration was also given to heavy metals and organics associated with particulate emissions from the incinerator operation. Fugitive dust emissions were not considered a significant contribution to the risk assessment, and are not discussed further here.

The magnitude of the emissions of concern was estimated by a combination of empirical observations from comparable facilities (See ORF, 1985; Abkovitz et al., 1984) and by engineering calculations (See Monenco, 1985; Dillon, 1984; Dillon, 1985). The empirical data and the calculation methods are described here for the following facility emission sources:

(1) Transportation

- Accidental spill

(2) Waste Handling and Storage

- Waste unloading and transfer
- Routine spills
- Drum opening and transfer
- Waste storage
- Tank farm fire

(3) Physical/Chemical Treatment

- Dissolved air flotation
- Emulsion breaking
- Cyanide treatment
- Sulfide treatment
- Mixing of incompatible wastes

(4) Incineration

- Controlled stack emissions
- Scrubber failure

C.1.2 ASSUMPTIONS FOR CALCULATIONS

The calculations for the emissions provided by the engineering consultants (Monenco, 1985) were developed based on a single component system, i.e. an individual chemical. In order to extend these single component system estimates to the multiple component system found in a waste mixture, Raoult's Law for ideal vapor-liquid equilibria was used to approximate the behavior of hazardous wastes mixtures.

Raoult's Law for multi-component mixtures is based on the following assumptions:

- the gas phase behaves as an ideal gas
- the liquid phase behaves as an ideal liquid
- the fugacity of the mixture is insensitive to variations in pressure

Raoult's Law is given by:

$$Y_i = \frac{x_i P_i}{P_o}$$

where Y_i = mole fraction of component i in the gas phase

x_i = mole fraction of component-i in the liquid phase

P_i = vapor pressure of component i, mm Hg

P_o = ambient pressure, mm Hg

While it is recognized that there is currently a lack of either empirical or theoretical correlations capable of accurately predicting the vapor-liquid equilibrium for multi-component mixtures of complex hydrocarbons, considering the uncertainty with which the composition of the waste streams is known, the error introduced in assuming Raoult's Law is considered to be relatively small. The effective partial pressure of each component of the waste mixture to be handled at the OWMC facility was thus approximated from the mole fraction of each component of the mixture. The mole fractions for each component were derived from the mass fractions presented in Appendix B, Selection of Surrogate Chemicals.

Calculation of the mole fraction in the waste stream requires a knowledge of the molecular weights of the other waste constituents. Given the varying composition of waste streams, it is difficult to make any such determination precisely. Based on consideration of the range of constituents expected to be present in the waste streams to be received at the OWMC facility, an average molecular weight for the components of the mixture other than the constituent of concern was assumed to be 150.

The mole fraction was then estimated as:

$$X = \frac{MF_x(MW_{av})}{MW_x}$$

where X = mole fraction of constituent, dimensionless

MF_x = mass fraction of the constituent, dimensionless

MW_x = molecular weight of the constituent, g/mol

MW_{av} = average molecular weight for the waste stream, g/mol

The average molecular weight is defined as:

$$MW_{av} = (MF_x)(MW_x) + (1 - MF_x)(150)$$

so that the mole fraction can now be defined as:

$$X = \frac{MF_x\ [(MF_x)(MW_x) + (1 - MF_x)(150)]}{MW_x}$$

The mole fractions calculated in this way are presented in Table C-1.

TABLE C-1

MOLE FRACTIONS FOR ESTIMATION OF EMISSION RATES

Chemical Constituents	Fugitive Emission Mole Fraction	Incinerator Emission Mole Fraction
Trichloroethylene	2.25E-02	1.13E-02
Perchloroethylene	0.00E+00	9.33E-03
Chloroform	1.06E-02	0.00E+00
1,1,1-Trichloroethane	3.79E-03	2.04E-02
Hydrazine	0.00E+00	1.58E-03
Acrylonitrile	2.85E-03	9.52E-04
Methylene Chloride	6.12E-03	1.78E-04
Pyridine	5.36E-03	5.36E-03
Benzene	3.89E-03	3.88E-03
Toluene	1.30E-02	1.57E-02
Phenol	8.05E-05	7.84E-03
Formaldehyde	5.19E-03	6.49E-03
Petroleum Distillates	6.91E-02	6.91E-02
Tetraethyl Lead	7.79E-07	7.79E-07
PCB	4.85E-04	4.94E-04
Ethanol	1.84E-01	1.74E-01
Styrene	4.04E-02	2.02E-02
Methyl ethyl ketone	7.01E-04	3.49E-04
Cresols	0.00E+00	2.09E-02

Other data inputs used in the calculation of emissions are summarized in Table C-2. The vapor pressures were estimated at different temperatures using the Antoine equation (Lyman, 1982). The vapor pressures at 10°C (annual average) were necessary for fugitive emission calculations from activities conducted outdoors, such as waste unloading, transfer and storage. Indoor activities such as drum opening and transfer were assumed to be conducted at 25°C, while emulsion breaking is carried out in a heated unit at a temperature of 70°C.

Another assumption used generically in the calculation of fugitive emissions was the efficiency of adsorption/scrubbing in the plant ventilation systems. Fugitive emissions from the facility will generally be recovered or captured by adsorption/scrubbing which removes the bulk of the contaminants. The removal efficiency depends on the properties of the contaminant, and, for the purposes of the risk assessment, was conservatively assumed to be 90%.

Finally, two scenarios were considered for the capacity and configuration of the facility: an initial throughput of 150 000 t/a of wastes operating with a single incinerator (of capacity 30 000 t/a); and the ultimate capacity of 300 000 t/a with two incinerators (with a combined capacity of 60 000 t/a). While it might at first be expected that the difference between the two scenarios would be a simple doubling of emissions in going from 150 000 t/a to 300 000 t/a, for the fugitive emissions this is not necessarily the case. Some of the sources contributing to fugitive emissions exhaust to the incinerator feed air, others vent to the atmosphere, either directly or via an adsorber. When the incinerator is out of operation for maintenance those sources that typically exhaust to the incinerator, vent to the atmosphere via

TABLE C-2

CHEMICAL PROPERTY DATA FOR ESTIMATION OF EMISSION RATES**

CHEMICAL CONSTITUENT	MOLEC WEIGHT (g/mol)	P_i* @ 10 C (mm-Hg)	P_i @ 25 C (mm-Hg)	P_i @ 70 C (mm-Hg)	DIFF IN AIR (cm^2/s)
Trichloroethylene	131.389	3.51E+01(2)	7.38E+01(2)	4.39E+02(2)	8.75E-02
Perchloroethylene	165.834	6.20E+00(1)	1.40E+01(4)	1.42E+02(2)	7.97E-02
Chloroform	119.378	9.82E+01(2)	1.93E+02(2)	9.81E+02(2)	8.88E-02
1,1,1-Trichloroethane	133.405	4.40E+01(1)	1.00E+02(5)	7.60E+02(3)	7.94E-02
Hydrazine	32.05	7.10E+00(1)	1.60E+01(5)	1.44E+02(2)	1.90E-01
Acrylonitrile	53.064	5.07E+01(2)	1.05E+02(2)	5.97E+02(2)	1.06E-01
Methylene chloride	84.933	2.29E+02(2)	4.33E+02(2)	1.99E+03(1)	1.04E-01
Pyridine	79.102	8.50E+00(2)	2.06E+01(2)	1.66E+02(2)	9.50E-02
Benzene	78.114	4.52E+01(2)	9.45E+01(2)	5.48E+02(2)	9.32E-02
Toluene	92.141	1.23E+01(2)	2.82E+01(2)	2.03E+02(2)	8.49E-02
Phenol	94.113	1.40E-01(1)	5.00E-01(5)	1.00E+01(3)	8.50E-02
Formaldehyde	30.026	2.35E+03(2)	3.83E+03(2)	1.25E+04(2)	1.70E-01
Petroleum distillates	130	1.78E+00(1)	4.00E+00(6)	N/A	7.57E-02
Tetraethyl lead	323.44	7.00E-02(1)	1.52E-01(5)	1.00E+01(3)	1.00E-02
PCB	328.4	3.43E-05(1)	7.71E-05(4)	1.37E-01(2)	4.00E-02
Ethanol	46.069	2.33E+01(2)	5.86E+01(2)	5.38E+02(2)	1.18E-01
Styrene	104.16	4.40E+00(1)	6.12E+01(2)	6.04E+00(2)	7.50E-02
Methyl ethyl ketone	72.1	4.52E+01(2)	1.00E+02(3)	5.53E+02(2)	8.94E-02
Cresols	108.13	3.23E-02(1)	1.75E-01(5)	5.00E+00(3)	8.10E-02

* P_i symbolizes Vapor Pressure

** References are in parentheses

(1) Calculated

(2) Reid and Prausnitz, 1977

(3) CRC, 1984

(4) Mabey et al., 1982

(5) Verscheuren, 1983

(6) GE Manual, 1980

an adsorber. If however, there are two incinerator units, the source is essentially eliminated, since one incinerator unit is expected to be operational at all times. The only exception to this would occur if during the scheduled shutdown of one incinerator, the second unit was obliged to undergo an unscheduled shutdown.

Conversely, for those emission sources which normally vent to an adsorber or directly to the atmosphere, the emission rate at 300 000 t/a throughput is simply double that at 150 000 t/a. To determine the impact of these different effects as a function of waste throughput, emission rates were estimated for waste throughputs of both 150 000 t/a and 300 000 t/a, as presented in the following sections.

C.2 WASTE TRANSPORTATION

Wastes will arrive at the facility in trucks of varying types and sizes ranging from pick-ups to large multi-axle vehicles. Wastes generally will be containerized in three ways: drums or other types of containers; dump trucks or luggers; and tankers (tank trucks or tank trailers).

For the purposes of estimating emissions, a transportation spill scenario was envisioned whereby a 30 tonne tank truck carrying a load of liquid waste is involved in a collision on the local access road. The collision results in tank rupture and release of the entire contents of the truck. The released liquid spreads across the road surface to form a pool from which the selected surrogate chemical, methylene chloride, is released to the atmosphere. Clearly, different circumstances prevailing at the time of the spill (e.g.,

nature of the tank rupture, configuration of road surface, shoulder or roadside ditches) will affect the behavior of the liquid pool. As a conservative representation of pool size, the geometry of the pool was assumed to be circular with the center of the circle coincident with the centerline of the road, and the diameter constrained by the width of the paved surface of the road (7.0 m). The emission rate was then based on the equation of Stiver and Mackay (1982):

$$Q = \frac{(K_g)\ (A)\ (P)\ (M)}{(R)\ (T)}$$

where Q = emission rate, g/s

K_g = the gas mass transfer coefficient, m/s

A = area of spill, m^2

P = vapor pressure of the chemical or mixture, Pa

M = molecular weight of compound, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

This equation was developed for spills of particular chemicals i.e. for a single component system. To apply this approach to the multiple component system found in a waste mixture, the equation was modified to include the mole fraction for each waste component as described in Section C.1.2. The modified emission rate equation then becomes:

$$Q_i = \frac{(Kg_i)(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

where Q_i = emission rate for species i, g/s

Kg_i = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure for species i, Pa

x_i = mole fraction for species i, dimensionless

M_i = molecular weight of species i, g/mol

R = gas constant, $(Pa.\ m^3)/(mol.K)$

T = temperature, K

Use of this equation requires the calculation of a mass transfer coefficient for the chemical of concern, which can be estimated using the equation of Mackay and Matsugu (1973):

$$Kg = 0.0048\ (U)^{0.78}\ (d)^{-0.11}\ Sc^{-0.67}$$

where Kg = mass transfer coefficient, m/s

U = windspeed, m/s

d = diameter of pool, m

Sc = Schmidt number, dimensionless

Schmidt numbers, which represent the effect of fluid friction on mass transfer, were approximated using the method of Shen (1982) based on molecular weight of the compound. Compounds with molecular weights less than 100 were assigned a $Sc^{-0.67}$ of 0.7, those with molecular weight from 100 to 200 were assigned a $Sc^{-0.67}$ of 0.6, and those with molecular weights greater than 200 were assigned a $Sc^{0.67}$ of 0.5. Values of wind speed and pool diameter were selected for particular spill scenarios.

It is important to recognize that the gas mass transfer coefficient is applicable to the prediction of emission rates of particular chemicals from spills. Mass transfer of a chemical in a complex solution or mixture is a more complicated process, which may involve transport across several phase boundaries. Additionally, transport may be controlled by other materials present in the mixture. However, since these complexities are extremely difficult to quantify for a waste stream and are, in any event, likely to reduce the value of the mass transfer coefficient, it was considered an appropriate and conservative assumption to use the gas mass transfer coefficient for the constituent of concern.

The emission rate calculated in this manner is sensitive to environmental parameters, specifically wind speed, which affects the mass transfer coefficient (Kg) and ambient temperature. For example, the emission rate at 0°C will be approximately three times lower than at 30°C. For the purposes of the risk assessment, emission rates were estimated conservatively by using a temperature of 25°C, considered typical of average summertime temperatures in Southern Ontario. A wind speed of 1 m/s was used to correspond to the atmospheric stability class F subsequently used to model atmospheric transport (see Gartner Lee Associates Limited, 1982 for discussion of atmospheric stability classes).

C.3 BULK WASTE UNLOADING AND TRANSFERS

Waste arriving at the facility will typically be unloaded through below-ground reception basins (unloading tanks). For the initial plant throughput of 150 000 t/a there will be 13 tank truck unloading stations receiving both

organic and inorganic wastes; nine for handling inorganic wastes and four for organic wastes. (Additional stations will be added when the plant is expanded to a throughput of 300 000 t/a). A below-ground reception tank equipped with a removable screened cage will be provided for each of the inorganic and two of the organic unloading stations for separating gross solids. For the purpose of calculating the emissions from the basins it was assumed that all wastes (except PCBs) will be unloaded through the reception basins. The reception basins will be equipped with covers or doors and an extraction fan connected to a vent exhaust system. In normal practice, the doors will be opened to 2 m^2 during unloading. Each extraction fan will be designed to maintain a negative pressure in the basin when the doors are closed, and an air velocity of 0.3 m/s in the basin opening when the doors are open. The exhausts from the fans will be ducted to a water scrubber/adsorber system before venting to the atmosphere at a height of 9 metres.

Four scenarios were considered:

- organic waste reception - normal operating conditions (all organics)
- organic waste reception - scrubber failure (methylene chloride as surrogate chemical)
- inorganic waste reception - normal operating conditions (hydrogen cyanide as surrogate chemical)
- inorganic waste reception - scrubber failure (hydrogen cyanide as surrogate chemical)

Emission calculations were performed both for normal operation of the scrubber/adsorber assuming 90% removal of air pollutants, and scrubber/adsorber failure where no pollutant removal was assumed (Monenco, 1985). The calculations are based on a capacity of 150 000 t/a which

represents the initial capacity of the facility under normal operation. At the ultimate capacity of 300 000 t/a, emissions under normal operating conditions will be doubled. Emissions under upset conditions were dealt with on a short term basis and will remain unaffected when plant capacity is increased.

C.3.1 ORGANIC WASTE RECEPTION - NORMAL OPERATION

Under normal operating conditions and a facility capacity of 150 000 t/a the emission rate for organic waste unloading and transfer is given by (Monenco, 1985):

Q_i = (molar flow rate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$Q_i = \frac{(V_1 T \dot{d})}{T_1} \frac{(x_i P_i)}{P_o} M_i (1-\eta) s N_o$$

where

Q_i	=	emission rate of species i, g/s
V_1	=	volumetric flow rate of air into basin per shipment, m^3/s
T_1	=	ambient temperature, K
T	=	standard condition temperature, K
d	=	molar density of air @ 273K and 760 mm Hg
x_i	=	mole fraction of species i in waste
P_i	=	vapor pressure of species i at T_1, mm Hg
P_o	=	ambient pressure, mm Hg
M_i	=	molecular weight of species i, g/mol
η	=	adsorber efficiency
s	=	percent saturation expressed in fraction
N_o	=	number of shipments

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- The cross-sectional area of the basin opening will be 2 m^2.
- Air flows in the basin opening at a rate of 0.3 m/s.
- Air leaving the basin will attain 20% saturation with the waste.
- Each shipment will require 15 minutes to unload and three shipments will be received per day for 250 days per year.
- The adsorber efficiency will be 90%.

$$V_l = (2\ m^2)(0.3\ m/s)\frac{(15\ min\ 60\ s/min)}{86\ 400\ s} = 0.0063\ m^3/s$$

T_l = 283K
T_s = 273K
d = 1/0.0224 g mol/m^3
P_o = 760 mm Hg
η = 0.9
s = 0.2
N_o = 3

Therefore, at 150 000 t/a plant capacity, on an average basis, the emission rate of species i (in g/s) is calculated as:

$$Q_i = \frac{(0.0063\ m^3/s)\ (273K)\ (x_i P_i)\ M_i\ (1-0.9)(0.2)(3)}{(283K)\ (0.0224\ m^3/g\ mol)\ (760\ mm\ Hg)}$$

$$= 2.1 \times 10^{-5}\ x_i P_i\ M_i$$

At a peak capacity of 300 000 t/a the emission rate will be doubled so that:

$$Q_i = 4.3 \times 10^{-5}\ x_i\ P_i\ M_i$$

C.3.2 ORGANIC WASTE RECEPTION - SCRUBBER/ADSORBER FAILURE

In the event the scrubber/adsorber fails, i.e., the removal efficiency becomes 0% ($\eta = 0$), and the emission rate due to one shipment becomes:

$$Q_i = \frac{(0.6\ m^3/s)\ (273K)\ (x_i P_i)\ M_i\ (1-0)\ (0.2)\ (1)}{(283K)\ (0.0224\ m^3/g\ mole)\ (760\ mm\ Hg)}$$

$$= 6.8 \times 10^{-3}\ x_i P_i\ M_i\ \ g/s$$

Using methylene chloride as the selected surrogate chemical:

$$Q_i = 0.81\ g/s\ methylene\ chloride$$

C.3.3 INORGANIC WASTE RECEPTION - NORMAL OPERATION

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for inorganic waste unloading and transfer is given by (Monenco, 1985):

Q_i = (molar gas flow rate) (adsorber efficiency factor)

$$Q_i = \frac{P_o V M (1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flow rate of HCN through basin per shipment, m^3/s

R = gas constant, m^3 mm Hg/mol/K

T = standard condition temperature, K

M = molecular weight of HCN

η = adsorber efficiency

Based on the facilty design and operations (Monenco, 1985), the following assumptions were then made for calculation of emissions:

- 50 shipments (800 t/a) of cyanide bearing sludge will be received per year giving a maximum of 1 shipment per day
- the absorber efficiency will be 90%

P_o = 760 mm Hg
V = 0.00114 m^3 of HCN/shipment/day at 273 K
= 0.00114 m^3/86 400 s
= 1.3 x 10^{-8} m^3/s
R = 0.0624 m^3 mm Hg/mol/K
T = 273 K
M = 27.03 g/mol
η = 0.9

Therefore, assuming a maximum of 1 shipment per day:

$$Q_i = \frac{(760 \text{ mm Hg})(1.3 \times 10^{-8} m^3/s)(27.03 \text{ g/mol})(1-0.9)}{(0.0624 \ m^3 \text{ mm Hg/mol/K})(273 \text{ K})}$$

$$= 1.6 \times 10^{-6} \text{ g/s hydrogen cyanide}$$

At the peak capacity of 300 000 t/a, the number of shipments becomes 100 shipments per year, so that the average shipment rate will still not exceed one shipment per day and the emission rate will be unchanged on a daily basis.

C.3.4 INORGANIC WASTE RECEPTION - SCRUBBER/ADSORBER FAILURE

In the event the scrubber/adsorber fails, i.e., the removal efficiency becomes 0% (η = 0), the emission rate due to one shipment becomes:

$$Q_i = \frac{(760 \text{ mm Hg})(1.27 \times 10^{-6} \ m^3/s)(27.03 \text{ g/mol})(1-0)}{(0.0624 \ m^3 \text{ mm Hg/mol/K})(273 \text{ K})}$$

$$= 1.52 \times 10^{-3} \text{ g/s}$$

C.3.5 ROUTINE SPILLS

Fugitive emissions may occur while unloading and transferring waste during hose coupling/uncoupling or other related transfer operations. The magnitude of these emissions was based on empirical data developed in a study of hazardous waste transportation spills in the United States (Abkowitz et al., 1984). Data were available from this study for the fraction of cargo lost during various stages in the transportation cycle without any indication of how the spilled chemical would subsequently be partitioned between air, surface water and groundwater.

To estimate the fraction of hazardous waste constituent volatilized to the atmosphere, the same model that was used for characterizing transportation releases in Section C.2 was applied:

$$Q_i = \frac{(Kg_i)(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

where Q_i = emission rate for species, g/s

Kg_i = the gas mass transfer coefficient for species, m/s

A = area of the spill, m^2

P_i = vapor pressure for species, Pa

x_i = mole fraction for species, dimensionless

M_i = molecular weight of species, g/mol

R = gas constant, Pa m^3/mol K

T = temperature, K

The total area of waste generated by routine spills (A) was estimated to be:

$$A = \frac{(W)(fw)}{(b)(\rho)}$$

where A = total spill area, m^2

W = daily quantity of organic waste transported by tanker, tons

fw = fraction of waste released

ρ = average density of wastes, kg/L

b = waste film thickness, m

Based on the facility design and operations, the following assumptions were made to approximate the area of spilled wastes available for volatilization:

- at 150 000 t/a plant capacity, 14 825 t/a organic waste will be transported by tanker (Monenco, 1985).
- the fraction of the waste released at terminal points, i.e. loading and unloading points, will be 8×10^{-6} at the OWMC facility (Abkowitz, M., et al., 1984). For routine spills on-site, the fraction of waste released at unloading points will be 4×10^{-6}.
- the spilled wastes will form a thin film assumed to have a thickness of approximately 0.001 m.
- average waste density is approximately 1.0 kg/L.

It was recognized that the assumptions regarding the thickness of the waste film is subject to considerable uncertainty. This assumption however will likely overestimate emissions since the concrete pad in the unloading

area will likely tend to absorb some fraction of spilled liquid. Similarly rainfall events will periodically remove such spillage to the facility drainage system, so that the spill area is conservatively estimated as:

$$A = \frac{(14\ 825\ t/a)(4 \times 10^{-6})(10^{3}\ kg/t)(10^{-3}\ m^{3}/L)}{(250\ d/a)(0.001\ m)(1.0\ kg/L)}$$

$$= 0.24\ m^{2}$$

Since these losses are transportation-related spillages that occur outside the confines of a building without the option for use of pollution control devices, the releases will occur directly to the atmosphere without any removal in an adsorber unit.

Therefore, at 150 000 t/a plant capacity the emission rate for routine spills is given by:

$$Q_i = \frac{(0.24)(Kg_i)(P_i)(M_i)}{(8.314)(283)}$$

$$= (1.02 \times 10^{-4})(Kg_i)(P_i)(M_i)$$

At a peak capcity of 300 000 t/a the emission rate will be doubled to:

$$Q_i = (2.04 \times 10^{-4})(Kg_i)P_i)(M_i)$$

To ensure that this emission rate does not exceed the available quantity of wastes, a mass balance was performed, and an emission rate calculated assuming that the quantity of wastes spilled per day would all be volatilized during the same day. The lower of the two emission rates was used for the purposes of the risk assessment.

C.4 DRUM UNLOADING AND TRANSFER

The drum storage and handling area will be designed to accept wastes arriving in drums up to 200 L in size. The area will contain four drum unloading stations for organic wastes, each consisting of a vacuum receiver, a suction pipe, a vacuum pump and a transfer pump. Drums will be opened by either removing the lid or screwed filling hole plug or by cutting a hole in the top and tranferring the contents to a receiving tank. The emission calculations were based conservatively on complete removal of the lid.

The unloading operation generates fugitive emissions in three ways: initial drawing of the vacuum in the receiver, maintenance of the vacuum while the wastes are pumped from the drums, and displacement of vapor in the tanks as they are filled. Fugitive vapors generated during drum opening will be collected by a ventilation system which exhausts the air through an activated carbon adsorber. Normal emissions are based on an adsorber efficiency of 90%. The emission from the three drum waste transfer operations will be exhausted from the vacuum pumps to a common header which vents to the incinerator. Initially, at 150 000 t/a capacity only one incinerator will be operational. When the incinerator is down, the vacuum pumps will exhaust to an activated carbon adsorber system. These drum transfer emissions were based on 90% efficiency of the carbon adsorption system during the 1760 hours the incinerator is out of service (Monenco, 1985).

At 300 000 t/a capacity there will be two incinerators operational with shut-down time staggered so that at least one incinerator will be functional

at all times. Hence, the fugitive emission during drum opening will double but the drum waste transfer emissions will be zero.

C.4.1 DRUM OPENING

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for drum opening is given by (Monenco, 1985); a modified form of the formula is:

$$Q_i = \frac{A \; D_{i,B} \; P \; (P_{i1} - P_{i2}) \; M_i \; (1-\eta)}{R \quad T \quad Z \quad P_{B,m}}$$

where

Q_i = emission rate, g/s

A = liquid surface area of all drums handled daily, cm^2

$D_{i,B}$ = diffusivity of species i in air @ 25°C, cm^2/s

P = ambient pressure, mm Hg

P_{i1} = partial pressure of species i @ gas-liquid interface; $P_{i1} = (P_i)(x_i)$

P_{i2} = partial pressure of species i, 5 cm from surface of liquid

$P_{B,m}$ = logarithmic mean of the partial pressures of gases other than i at the waste-air interface and 5 cm from the surface of liquid

R = gas constant, $(cm^3.mm\ Hg)(g\ mol.K)$

M_i = molecular weight of species i

T = ambient temperature, K

x_i = mole fraction of i in wastes

Z = diffusion distance, cm

η = adsorber efficiency

P_i = vapor pressure of species i

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- The adsorber efficiency is 90%.
- A safety factor of 3 times the calculated diffusion rate was incorporated (Monenco, 1985).
- 10 drums, each of cross-sectional area 2420 cm^2, will be open for 7 h per day.

$A = 24200\ cm^2$

$P = 760\ mm\ Hg$

$P_{i2} = 0\ mm\ Hg$

$$P_{B,m} = \frac{(760-0) - (760-x_iP_i)}{\ln \frac{(760-0)}{(760-x_iP_i)}} = \frac{x_iP_i}{\ln[760/(760-x_iP_i)]}$$

$R = 62400(cm^3.mm\ Hg)/(g\ mol.K)$

$T = 298\ K$

$Z = 5\ cm$

$\eta = 0.9$

Therefore, at 150 000 t/a plant capacity, on a daily average basis:

$$Q_i = \frac{3(24200cm^2)(D_{i,B})(760\ mm\ Hg)x_iP_iM_i(7h)(1-0.9)\ln[760/(760-x_iP_i)]}{62400(cm^3.mm\ Hg)/(g\ mol.K)\ (298\ K)\ (5\ cm)\ (24\ h)\ (x_iP_i)}$$

$$= 0.0173\ D_{i,B}\ M_i\ \ln\ [760/(760-x_iP_i)]$$

At the peak capacity of 300 000 t/a the emission rate will be doubled so that:

$$Q_i = 0.0346\ D_{i,B}\ M_i\ \ln\ [760/(760-x_iP_i)]$$

C.4.2 DRUM TRANSFER

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate via drum transfer is the summation of the emission rates due to:

- establishment of initial evacuation
- maintenance of vacuum
- displacement of vapor while filling tank

The contribution of each of these emissions was calculated as follows:

a) Establishment of initial evacuation

The emission rate for initial evacuation of the receiving tank is given by (Monenco, 1985):

Q_{ai} = (molar flowrate of air)(mole fraction of species in gas phase) (adsorber efficiency factor)

$$= \frac{(P_1 - P_2)\ V}{RT} \quad \frac{(x_i P_i)\ M_i\ (1-\eta)}{P_1}$$

where Q_{ai} = emission rate, g/s

P_1 = initial system pressure mm Hg

P_2 = final system pressure, mm Hg

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

T = ambient temperature, K

V = volumetric flowrate

P_i = vapor pressure of species i @ T

M_i = molecular weight of i

x_i = mole fraction of i in liquid phase

η = adsorber efficiency

Based on the facility design and operations (Monenco, 1985) , the following assumptions were made for calculation of emissions:

- assume 90% efficiency of adsorber ($\eta = 0.9$)
- volumetric flowrate at 150 000 t/a initial capacity would be 32 m^3/day

$$V = \frac{32\ m^3}{86\ 400\ s} = 3.7 \times 10^{-4}\ m^3/s$$

P_1 = 760 mm Hg
P_2 = 400 mm Hg
R = 0.0624 (m^3.mm Hg)/(mol.K)
T = 298 K

Therefore, at 150 000 t/a plant capacity:

$$Q_{ai} = \frac{(760\text{-}400\ \text{mm Hg})(3.7 \times 10^{-4}\ m^3/s)P_i x_i M_i (1\text{-}0.9)}{0.0624\ (m^3.\text{mm Hg})(\text{mol.K})\ (298\ \text{K})\ (760\ \text{mm Hg})}$$

$$= 9.42 \times 10^{-7}\ P_i\ x_i\ M_i$$

b) Maintenance of vacuum

The emission rate for maintenance of pressure is given by (Monenco, 1985):

Q_{bi} = (molar flowrate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$Q_{bi} = \frac{(PV)\ (x_i P_i) M_i\ (1\text{-}\eta)}{RT\ \ P_o}$$

where

Q_{bi} = emission rate, g/s

P = standard condition pressure, mm Hg

V = volumetric leak rate at standard condition, m^3/s

T = standard condition temperature, K

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

P_o = system pressure, mm Hg

x_i = mole fraction of species in waste

η = adsorption efficiency

P_i = vapor pressure of species i @ T, mm Hg

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- assume 90% efficiency of adsorber ($\eta = 0.9$)
- assume 2 systems, each leaking @ 2.5 m^3/h for 7 h/d

P_s = 760 mm Hg
V = 2 (2.5 m^3/h) (7 h/d) (1 day/8.64 x 10^4s)
= 4.07 x 10^{-4} m^3/s
T = 298 K
R = 0.0624 $(m^3.mm\ Hg)/(mol.K)$
P = 400 mm Hg

Therefore, at 150 000 t/a plant capacity:

$$Q_{bi} = \frac{(760\ mm\ Hg) \times (4.07 \times 10^{-4}\ m^3/s) P_i\ x_i\ M_i\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)(298\ K)\ (400\ mm\ Hg)}$$

$$= 4.1 \times 10^{-6}\ P_i\ x_i\ M_i$$

c) Displacement of vapor while filling tank

The emission rate of displaced vapor while filling the tank is given by (Monenco, 1985):

Q_{ci} = (molar flowrate of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$= \frac{(P_o V)}{RT} \frac{(P_i\ x_i\ M_i)}{P_o} (1-\eta)$$

$$Q_{ci} = \frac{V\ P_i\ x_i\ M_i\ (1-\eta)}{RT}$$

where

Q_{ci} = emission rate, g/s

V = volumetric flowrate, m^3/s

T = system temperature, K

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

P_o = system pressure, mm Hg

x_i = mole fraction of waste species i in waste

η = adsorption efficiency

P_i = vapor pressure of species i @ T

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 90% efficiency of adsorber ($\eta = 0.9$)
- 80% of the 2 systems (volume $16m^3$ each) will be evacuated per day

$$V = \frac{(0.8)\ (2 \times 16\ m^3)}{(86400\ s)} = 2.96 \times 10^{-4}\ m^3/s$$

$R = 0.0624\ (m^3.mm\ Hg)/(mol.K)$
$T = 298\ K$

Therefore, at 150 000 t/a plant capacity:

$$Q_{ci} = \frac{(2.96 \times 10^{-4}\ m^3/s)\ P_i\ x_i\ M_i\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)\ (298)}$$

$$= 1.59 \times 10^{-6}\ P_i\ x_i\ M_i$$

Hence, summing Q_{ai}, Q_{bi}, and Q_{ci}, the total emission rate via drum transfer at 150 000 t/a plant capacity, is given by:

$$Q_{Ti} = Q_{ai} + Q_{bi} + Q_{ci}$$

$$= 6.63 \times 10^{-6}\ x_i\ P_i\ M_i$$

At 300 000 t/a the vapor will be vented through the incinerators at all times, so that the fugitive emissions from drum transfer will become zero.

C.5 WASTE STORAGE TANKS

The waste storage tanks will be used for storage, blending and pre-treatment of pumpable wastes received from waste generators and from on-site waste sources. Fugitive emissions of working losses will result from the displacement of vapors from the storage tanks as the tanks are being filled.

For purposes of the risk assessment, three scenarios were considered:

- organic waste storage - normal operating conditions (all organics)
- inorganic waste storage - normal operating conditions (hydrogen cyanide as surrogate chemical)
- tank farm fire - accidental release (methylene chloride as surrogate chemical).

At 150 000 t/a capacity, under normal operating conditions, the displaced vapors from the organic waste storage will be vented to the incinerator, except for 1760 hours, when the incinerator is out of operation and the vapors will be vented through an activated carbon adsorber. The emission rates for fugitive emissions were calculated for a day when the incinerator is out of operation assuming an adsorber efficiency of 90%. At 300 000 t/a plant capacity fugitive emissions will be eliminated by the presence of a second incinerator.

In the case of the tank farm fire, the emission rate was based on the entire contents of a tank containing the selected surrogate chemical, methylene chloride (see Appendix B) boiling off as a result of the tank being surrounded by flames.

C.5.1 ORGANIC WASTE STORAGE

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for organic waste storage, working losses is given by (Monenco, 1985):

Q_i = (moles of air) (mole fraction of species in gas phase) (adsorber efficiency factor)

$$= \frac{(P_o V)}{RT} \frac{(x_i P_i M_i)}{P_o} (1-\eta) N_o = \frac{V x_i P_i M_i (1-\eta) N_o}{RT}$$

where

Q_i = emission rate, g/s

P_o = pressure of system, mm Hg

R = gas constant, = 0.0624 (m^3.mm Hg)/(mol.K)

T = temperature of tank, K

V = volume of shipment

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i @ T

M_i = molecular weight of species i

N_o = number of shipments per day

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 100% saturation of air with species i is attained
- volume of shipment is 15 m^3
- 90% efficiency of adsorber (η = 0.9)
- 5 shipments arrive per day

T = 283 K
V = 15 m^3
N_o = 5 shipments/86400s = 5.79 x 10^{-5}/s

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(15\ m^3)\ P_i\ x_i\ M_i\ (5.79 \times 10^{-5}/s)\ (1-\eta)}{0.0624\ (m^3.mm\ Hg)/(mol.K)(283\ K)}$$

$$= 4.91 \times 10^{-6}\ x_i P_i M_i$$

At a peak capacity of 300 000 t/a the displaced vapors will be routed to the incinerator feed air at all times, so that the emission rate becomes zero.

C.5.2 INORGANIC WASTE STORAGE

Under normal operating conditions and a plant capacity of 150 000 t/a, the emission rate for inorganic waste storage working losses is given by (Monenco, 1985):

$$Q_i = \frac{P_o V M_{HCN} (1 - \eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flow rate of contaminant, m^3/s at standard conditions

R = gas constant, $(m^3.mm\ Hg)/(mol.K)$

T = standard condition temperature, K

M = molecular weight of HCN

η = adsorber efficiency

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- the volumetric flow rate is 0.00165 m^3/a
- the adsorber efficiency is 90 %

P_o = 760 mm Hg
V = 0.00165 Nm^3 of HCN per year at 273 K
= 0.00165 Nm^3/(86 400) (250d)
= 7.65 x 10^{-11} Nm^3/s
R = 0.0624 $(m^3.mm\ Hg)/(mol.K)$
T = 273 K
M = 27.03 g/mol
η = 0.9

Therefore,

$$Q_i = \frac{(760 \text{ mm Hg})(7.65 \times 10^{-11} \text{ Nm}^3/\text{s})(27.03 \text{ g/mol})(1-0.9)}{0.0624 \text{ (m}^3\text{.mm Hg)/(mol.K)(273 K)}}$$

$$= 9.0 \times 10^{-9} \text{ g/s}$$

At a peak capacity of 300 000 t/a displaced vapors will be doubled so that:

$$Q_i = 1.8 \times 10^{-8} \text{ g/s}$$

C.5.3 TANK FARM FIRE

Tank farm fire emissions are also based on a conservative scenario developed in earlier work for OWMC in the selection of a buffer zone (Dillon, 1984), which subsequently was adopted by the engineering consultants (Monenco, 1985). In this scenario it is envisioned that a $10m^3$ (2500 gallon) tank full of the selected surrogate chemical methylene chloride is exposed to a fire surrounding the tank. The contents of the tank are boiled off and vented above the level of flame effect. Assuming that the tanks are uninsulated, the rate of boil-off, or the emission rate, was estimated by the method of the American Petroleum Institute (API, 1976) as:

$$Q_i = \frac{2652 \text{ (F)}(A_w)^{0.82}}{\Delta_H}$$

where

Q_i = emission rate, g/s

F = insulation factor = 1.0 for a bare vessel

A_w = wetted surface area of tank, ft^2

Δ_H = heat of vaporization 139 Btu/lb

The following assumptions were made (Dillon, 1984):

- the wetted surface area of the tank is 176.7 ft^2
- the tank capacity is 2 500 gallons

Therefore,

$$Q_i = \frac{2652\ (1)(176.7\ ft^2)^{0.82}}{(139\ BTU/lb)}$$

$$= 1328\ g/s$$

C.6 PHYSICAL CHEMICAL TREATMENT

The inorganic wastes will undergo preliminary physical chemical treatment which could be a source of fugitive emission. The following unit operations will be used in the treatment:

- dissolved air flotation and emulsion breaking to separate hydrocarbons from aqueous phase.
- oxidation/reduction reactions for the destruction of cyanides and reduction of chromium,
- neutralization/precipitation reactions for heavy metals removal.

Estimates were made for fugitive emissions arising from these sources assuming 150 000 t/a plant capacity. Additionally, emissions resulting from the mixing of incompatible wastes were estimated.

C.6.1 DISSOLVED AIR FLOTATION (DAF) UNIT

The DAF unit will be employed in the separation of suspended oil from aqueous solutions. The air above the liquid is first pressurized thereby increasing the solubility of air in the liquid. The pressure is then quickly released causing nucleation of air bubbles in the aqueous liquid which bubble to the surface. As the air bubbles rise through the waste liquid, globules of hydrocarbon adhere to the bubbles and are carried to the surface. The end result is that liquid hydrocarbon will tend to collect on the surface of the aqueous waste, where, due to the action of the air a layer of froth will be formed. The emission calculation assumes that the air is saturated with the hydrocarbon in the froth. This air is then released directly to the atmosphere.

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the DAF unit is given by (Monenco, 1985):

Q_i = (molar flowrate of air) (mole fraction of species in gas phase)

$$= \frac{(P_s V)}{RT} \frac{(x_i P_i) M_i}{P_o} = \frac{V x_i P_i M_i}{RT}$$

where

Q_i = emission rate, g/s

P = ambient pressure, mm Hg

R = gas constant, 0.0624 $(m^3.mm\ Hg)/(mol.K)$

T = standard condition temperature (273 K)

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i @ 25°C

M_i = molecular weight of species i

V = volumetric flowrate of air at standard conditions, m^3/s

P_s = pressure at standard conditions, ~ P_o

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- the unit will be in operation 19 hours per day on an average basis
- the volumetric flowrate is 0.003 Nm^3/min

$$V = (0.003\ Nm^3/min)(min/60\ s)\ \frac{(19\ h)}{(24\ h)} = 3.96 \times 10^{-5}\ m^3/s$$

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(3.96 \times 10^{-5}\ m^3/s)\ x_i\ P_i\ M_i}{0.0624\ (m^3.mm\ Hg)/(mol.K)(273)}$$

$$= 2.3 \times 10^{-6}\ x_i\ P_i\ M_i$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 4.6 \times 10^{-6}\ x_i\ P_i\ M_i$$

C.6.2 EMULSION BREAKING

The oil (hydrocarbon)/water emulsions will be broken by heating. As the emulsion-breaking tank is filled, vapor will be displaced and vented through an adsorber/scrubber system. Air in equilibrium with hydrocarbon/oil will be vented through an adsorber which is assumed to have an efficiency of 90%.

The following emission calculations are based on a 150 000 t/a plant capacity. The estimates would double at the ultimate capacity of 300 000 t/a. Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the emulsion-breaker is given by (Monenco, 1984):

$$Q_i = \frac{(P_o V)}{RT} \frac{(x_i P_i)}{P_o} M_i(1-\eta) = \frac{V\, x_i\, P_i\, M_i(1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = pressure of system, 760 mm Hg

T = temperature of gas, 343 K

R = gas constant = 0.0624 $(m^3.mm\ Hg)/(mol.K)$

x_i = mole fraction of species i in waste

P_i = vapor pressure of species i at T

M_i = molecular weight of species i

V = volume expelled per second

Based on the facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 2 batches per day each of 25 m^3
- 90% adsorber efficiency (η=0.9)

$$V = 2\ (25\ m^3/d)(d/8.64 \times 10^4\ s)$$

$$= 5.79 \times 10^{-4}\ m^3/s$$

Thus, the emission rate at 150 000 t/a can be calculated as:

$$Q_i = \frac{(5.79 \times 10^{-4}\ m^3/s)\ M_i\ x_i\ P_i\ (1-0.9)}{0.0624(m^3.mm\ Hg)/(mol.K)\ (343\ K)}$$

$$= 2.70 \times 10^{-6}\ x_i\ P_i\ M_i$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 5.4 \times 10^{-6}\ x_i\ P_i\ M_i$$

C.6.3 CYANIDE TREATMENT

The cyanide bearing waste will be destroyed by the addition of an oxidizing agent such as hypochlorite or hydrogen peroxide. Air from the unit will be vented through the scrubber/adsorber. For calculation of emissions under normal operating conditions, it was assumed that the air leaving the unit will be saturated with hydrogen cyanide and vented through a scrubber with an efficiency of 90%.

The situation was also analyzed under upset conditions. Under abnormal conditions, i.e., when the pH control systems fails, the saturation concentration of hydrogen cyanide in equilibrium with the solution increases while the absorber efficiency remains at 90%. It was assumed that the situation would be brought under control after a brief interval of time.

C.6.3.1 Normal Operations

Under normal operating conditions and a facility capacity of 150 000 t/a, the hydrogen cyanide emission rate is given by (Monenco, 1984):

$$Q_i = \frac{P_o V M_{HCN} (1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flowrate at standard condition

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature, 273 K

η = adsorber efficiency

M_{HCN} = molecular weight of HCN, g/mol

Based on facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions:

- 200 batches are treated per year
- 1 batch treated per day evolves 0.0011 m^3 of vapor
- scrubber/adsorber efficiency is 90% (η = 0.9)
- M_{HCN} is 27.03 g/mol

P = 760 mm Hg

$$V = \frac{0.0011\ m^3}{86400\ s} = 1.273 \times 10^{-8}\ m^3/s$$

Therefore, at 150 000 t/a:

$$Q_i = \frac{(760 \text{ mm Hg})\ (1.273 \times 10^{-8} \text{ m}^3/\text{s})\ (27.03 \text{ g})\ (1-0.9)}{0.0624\ (\text{m}^3.\text{mm Hg})/(\text{mol.K})\ (273)}$$

$$= 1.5 \times 10^{-6} \text{ g/s}$$

At the peak capacity of 300 000 t/a the emissions would double to 3.1×10^{-6} g/s.

C.6.3.2 Uncontrolled pH

Under abnormal conditions instead of a normal pH of 9.5, a pH reduction to 7.5 would result in a higher HCN vapor concentration in the gas. Vapor emission over a 6 hour period would be:

$$V = \frac{0.053 \text{ m}^3}{6 \times 3600 \text{ s}} = 2.45 \times 10^{-6} \text{ m}^3/\text{s}$$

Therefore, the emission rate would be:

$$Q_i = \frac{(760 \text{ mm Hg})\ (2.45 \times 10^{-6} \text{ m}^3/\text{s})\ (27.03 \text{ g/mol})\ (1-0.9)}{0.0624\ (\text{m}^3.\text{mm Hg})/(\text{mol.K})\ (273 \text{ K})}$$

$$= 3.0 \times 10^{-4} \text{ g/s of hydrogen cyanide}$$

C.6.4 SULFIDE TREATMENT

The removal of heavy metals from waste solutions will be accomplished by the addition of a sodium sulfide solution to a batch of the waste. In the

alkaline state, this results in the precipitation of heavy metals as sulfides, and emission of hydrogen sulfide is negligible.

If, during abnormal conditions, the pH is allowed to fall to the acidic state the concentration of gaseous hydrogen sulfide in equilibrium with the waste solution increases. The emission calculation is based on such a failure with saturated air venting through a water scrubber with an efficiency of 90%.

Under abnormal conditions and a facility capacity of 150 000 t/a, the emission rate of hydrogen sulfide is given by (Monenco, 1985):

$$Q_i = \frac{P_o V M_{H2S} (1-\eta)}{RT}$$

where

Q_i = emission rate, g/s

P_o = standard condition pressure (760 mm Hg)

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature (273 K)

V = volumetric flowrate of gas at standard condition

η = scrubber/adsorber efficiency

M_{H2S} = molecular weight, 34.08 g/mol

Based on facility design and operations (Monenco, 1985), the following assumptions were made for calculation of emissions under abnormal pH situation:

- the adsorber efficiency is 90% (η=0.9)
- the volumetric flowrate of gas is 0.013 m^3/hr
- pH falls to 5

$$V = (0.013\ m^3/h)\ (h/3600\ s)$$

$$= 3.61 \times 10^{-6}\ m^3/s$$

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(760\ mm\ Hg)\ (3.61 \times 10^{-6}\ m^3/s)\ (34.08\ g)\ (1-0.9)}{0.0624\ (m^3.mm\ Hg)/(mol.K)\ (273\ K)}$$

$$= 5.49 \times 10^{-4}\ g/s$$

C.6.5 MIXING OF INCOMPATIBLE WASTES

An accidental release considered in the risk assessment was the generation of hydrogen sulfide as a result of mixing of incompatible wastes. The scenario considered was developed in earlier work for OWMC on the selection of a technical buffer zone (M.M. Dillon, 1984).

In this scenario it was envisioned that 4% sulfuric acid was mixed with sulfide contaminated material in the physical/chemical treatment building. The gas is emitted first into the building and then via its ventilation system to the atmosphere. The situation was simulated in the laboratory on a reduced scale by mixing 4% sulfuric acid with zinc sulfide. Based on the laboratory data the emission rate for this scenario over a 30 minute interval was estimated to be 0.462 g/s (Dillon, 1984).

C.7 INCINERATION

An incinerator will be used to destroy organic wastes at high temperatures. Flue gases from the incinerator will be cleaned by a flue gas treatment system consisting of a spray dryer, an electrostatic precipitator, an induced draft

fan and a three stage wet gas scrubber. The incinerator operations will result in air pollutants generated from a number of sources:

- organic chemicals in feed,
- acid gases generated by combustion,
- metals present in the waste feed,
- products of incomplete combustion

Estimates were made for these controlled stack emissions and for emissions under upset conditions in the event of scrubber failure.

C.7.1 CONTROLLED STACK EMISSIONS

Estimation of controlled stack emission rates were based on two approaches: a design destruction efficiency in the case of emissions of residual organics, and empirical data for metals, acid gases and dioxins. These approaches are described in the following sections.

C.7.1.1. Incinerator Stack Emissions - Organics

Under normal operating conditions, and a facility capacity of 150 000 t/a, based on 99.99% DRE the emission rate for organics is given by (Monenco, 1985):

$$Q_i = Q_1 f_1 f_2$$

where

Q_i = emission rate of uncombusted chemical, g/s

Q_1 = waste throughput, g/s

f_1 = fraction of waste that represents constituent of concern

f_2 = fraction of waste uncombusted based on 99.99% DRE

Based on the facility design and operating conditions (Monenco, 1985), the following assumptions were made for calculation of emissions:

- DRE of 99.99% implies $f_2 = 10^{-4}$
- annual throughput = 30 000 metric tons (952.2 g/s)

Therefore, at 150 000 t/a plant capacity:

$$Q_i = (952.2) f_1 (1 \times 10^{-4})$$
$$= 0.0952\ f_1$$

At the peak capacity of 300 000 t/a, two incinerators each operating at 30 000 t/a is envisioned; resulting in an emission rate of:

$$Q_i = 0.19\ f_1$$

C.7.1.2 Incinerator Stack Emission-Metals

The presence of metals in the wastes results in metals emissions to the atmosphere. Many metals evaporate in the flame, either as such, or in the form of volatile oxides, chlorides or other salts. A small percentage of the metals escapes the incinerator emission controls and vents to the atmosphere. Based on comparative facilities and OWMC facility design information the following are estimates of controlled emissions (Q) arising from metals in the feed to an incinerator (ORF, 1985).

TABLE C-3

STACK EMISSION ESTIMATES - METALS

Metal	Emission Rate @ 150 000 t/a	Emission Rate @ 300 000 t/a
Arsenic	4.2×10^{-5} g/s	8.3×10^{-5} g/s
Beryllium	2.8×10^{-7} g/s	5.6×10^{-7} g/s
Cadmium	4.2×10^{-4} g/s	8.3×10^{-4} g/s
Chromium	9.7×10^{-4} g/s	1.9×10^{-3} g/s
Copper	9.0×10^{-4} g/s	1.8×10^{-3} g/s
Lead	4.7×10^{-3} g/s	9.4×10^{-3} g/s
Mercury	3.5×10^{-4} g/s	6.9×10^{-4} g/s
Nickel	4.2×10^{-4} g/s	8.3×10^{-4} g/s
Zinc	4.9×10^{-3} g/s	9.7×10^{-3} g/s

C.7.1.3 Incinerator Stack Emissions-Acid Gases

Emissions of acid gases will be dependent on the waste composition, the incinerator design and operations, and the gaseous and particulate control equipment. The emission (Q) data presented in the following table for plant capacities of 150 000 t/a and 300 000 t/a were determined from data in the literature for comparable facilities and OWMC facility design information (ORF, 1985).

TABLE C-4

STACK EMISSION ESTIMATES - ACID GASES

Acid Gas	Q @ 150 000 t/a	Q @ 300 000 t/a
HCl	0.28 g/s	0.6 g/s
HF	6.9×10^{-3} g/s	1.4×10^{-2} g/s
SO_2	1.04 g/s	2.07 g/s
NO_x	1.04 g/s	2.07 g/s

C.7.1.4 Incinerator Stack Emissions - Products of Incomplete Combustion

Products of incomplete combusion (PICs) are formed as a result of partial oxidation of chemicals in the waste feed due to the combination of molecular fragments outside of the combustion zone. At present there is limited information on the mechanism by which these PICs are formed, and how their formation can be related to waste feed or incinerator operating conditions (ORF, 1985). In the absence of such information, consideration was given to a particular set of possible PICs: the dioxins and furans. Based on comparative facilities and OWMC facility design information the following are estimates of controlled emissions PICs from the incinerator (ORF, 1985).

TABLE C-5

STACK EMISSION ESTIMATES - PICs

PICs	Emission Rate @ 150 000 t/a	Emission Rate @ 300 000 t/a
Dioxins	1.5×10^{-7} g/s	3.1×10^{-7} g/s
Furans	7.1×10^{-7} g/s	1.4×10^{-6} g/s

C.7.2 INCINERATOR SCRUBBER FAILURE

In the event of a flue gas scrubber failure, the emergency power will be designed to resume operations in less than 2 minutes (Monenco, 1985). Gas flow would decrease to 10% of normal in 4 minutes following failure. In the calculation of emission rates it was assumed that at 150 000 t/a facility capacity one incinerator would be in operation and at 300 000 t/a, two incinerators would be operational. Each incinerator has a capacity of about 30 000 t/a. Based on data supplied by Monenco and the above scenario the emission rates for the two minutes of failure will be:

Q_i = (Average volumetric flow rate) (Acid gas concentrations)

$= (V)(C_g)$

where

Q_i = emission rate, g/s

V = average volumetric flowrate, 5.5 m^3/s

C_g = acid gas concentrations, g/m^3

The emission rates at 150 000 t/a and 300 000 t/a facility capacity are presented in Table C-6. At 300 000 t/a, it was conservatively assumed that power failure would result in scrubber failure for both incinerator units.

TABLE C-6

STACK EMISSION ESTIMATES - SCRUBBER FAILURE

Acid Gases	Acid Gas Concentrations (g/m^3)	Emission Rate @ 150 000 t/a (g/s)	Emission Rate @ 300 000 t/a (g/s)
HCl	12.618	69.0	138.0
SO_2	9.787	53.5	107.0
HF	0.288	1.6	3.1
NO_x	0.285	1.6	3.1

C.8 SUMMARY OF RESULTS

The emission rates for the facility releases described in earlier sections are summarized in Tables C-7, C-8, C-9 and C-10.

Fugitive emissions for organic chemicals are presented in Tables C-7 and C-8 for plant capacities of 150 000 t/a and 300 000 t/a respectively. A comparison of these emissions data shows that the emissions for waste storage and drum transfer, become zero at 300 000 t/a capacity, while the other emissions are each doubled. The net effect of these changes is for the total fugitive emissions to increase slightly at the peak capacity.

TABLE C-7

FUGITIVE EMISSION RATES (g/s)

AT 150 000 t/a

Chemical Constituent	Waste Unload & Transfer	Drum Opening	Drum Transfer	Storage Tanks	DAF Unit	Emulsion Breaking	Routine Spills	Total Fugitive Emissions
Trichloroethylene	2.21E-03	4.36E-04	1.45E-03	5.10E-04	1.35E-05	9.26E-05	1.09E-04	4.82E-03
Chloroform	2.64E-03	4.94E-04	1.62E-03	6.08E-04			4.62E-05	5.40E-03
1,1,1-Trichloroethane	4.74E-04	9.14E-05	3.35E-04	1.09E-04	3.57E-05	3.12E-04	1.85E-05	1.38E-03
Acrylonitrile	1.63E-04	3.84E-05	1.05E-04	3.77E-05			5.54E-06	3.50E-04
Methylene Chloride	2.54E-03	5.34E-04	1.49E-03	5.85E-04			1.91E-05	5.17E-03
Pyridine	7.67E-05	1.88E-05	5.78E-05	1.77E-05			1.55E-05	1.87E-04
Benzene	2.93E-04	6.10E-05	1.91E-04	6.75E-05	5.26E-06	3.51E-05	1.11E-05	6.63E-04
Toluene	3.14E-04	6.53E-05	2.24E-04	7.24E-05			4.39E-05	7.19E-04
Phenol	2.26E-08	7.33E-09	2.51E-08	5.21E-09			5.43E-08	1.15E-07
Formaldehyde	7.81E-03	2.34E-03	3.96E-03	1.80E-03			5.71E-06	1.59E-02
Petroleum Distillates	3.40E-04	6.20E-05	2.38E-04	7.84E-05			3.32E-04	1.05E-03
Tetraethyl Lead	3.76E-10	8.60E-12	2.51E-10	8.66E-11			6.46E-10	1.37E-09
PCB		1.12E-11	8.14E-11	2.68E-11			2.00E-10	3.19E-10
Ethanol	4.20E-03	1.34E-03	3.29E-03	9.68E-03			3.23E-04	1.01E-02
Styrene	3.94E-04	4.40E-05	1.71E-04	9.09E-05			1.55E-04	8.55E-04
Methyl Ethyl Ketone	4.86E-05	1.03E-05	3.35E-05	1.12E-05			1.85E-06	1.05E-04

TABLE C-8

FUGITIVE EMISSION RATES (g/s)

AT 300 000 t/a

Chemical Constituent	Waste Unload & Transfer	Drum Opening	DAF Unit	Emulsion Breaking	Routine Spills	Total Fugitive Emissions
Trichloroethylene	4.43E-03	8.71E-04	2.70E-05	1.85E-04	2.17E-04	5.73E-03
Chloroform	5.27E-03	9.87E-04			9.25E-05	6.35E-03
1,1,1-Trichloroethane	9.47E-04	1.83E-04	8.71E-05	6.24E-04	3.70E-05	1.88E-03
Acrylonitrile	3.27E-04	7.68E-05			1.11E-05	4.15E-04
Methylene Chloride	5.08E-03	1.07E-03			3.81E-05	6.18E-03
Pyridine	1.53E-04	3.77E-05			3.11E-05	2.22E-04
Benzene	5.85E-04	1.22E-04	1.05E-05	7.02E-05	2.23E-05	8.10E-04
Toluene	6.28E-04	1.31E-04			8.78E-05	8.46E-04
Phenol	4.52E-08	1.47E-08			1.09E-07	1.69E-07
Formaldehyde	1.56E-02	4.68E-03			1.14E-05	2.03E-02
Petroleum Distillates	6.81E-04	1.24E-04			6.63E-04	1.47E-03
Tetraethyl Lead	7.51E-10	1.72E-11			1.29E-09	2.06E-09
PCB		2.24E-11			4.00E-10	4.22E-10
Ethanol	8.40E-03	2.68E-03			6.46E-04	1.17E-02
Styrene	7.89E-04	8.80E-05			3.11E-04	3.99E-04
Methyl Ethyl Ketone	$9.72E \times 10^{-5}$	2.06E-05			3.70E-06	1.22E-04

TABLE C-9

INCINERATOR STACK EMISSION ESTIMATES

AT 300 000 t/a

Chemical Constituent	Emission Rate (g/s)	Chemical Constituent	Emission Rate (g/s)
VOLATILE ORGANICS		ACID GASES	
Trichloroethylene	1.88E-03	Sulfur Dioxide	2.07
Perchloroethylene	1.96E-03	Nitrogen Oxides	2.07
Chloroform	0.00E-00	Hydrogen Chloride	0.56
1,1,1-Trichloroethane	3.46E-03	Hydrogen Fluoride	0.014
Hydrazine	6.42E-05		
Acrylonitrile	6.42E-05	METALS	
Methylene Chloride	1.92E-05		
Pyridine	5.39E-04	Lead	9.4E-03
Benzene	3.85E-04	Zinc	9.7E-03
Toluene	1.84E-03	Nickel	8.3E-04
Phenol	9.39E-04	Chromium	1.9E-03
Formaldehyde	2.48E-04	Arsenic	8.3E-05
Petroleum Distillates	1.15E-02	Cadmium	8.3E-04
Tetraethyl Lead	3.20E-07	Mercury	6.9E-04
PCB	2.06E-04	Copper	1.8E-03
Ethanol	1.06E-02	Beryllium	5.6E-07
Styrene	2.68E-03	DIOXINS/FURANS	
Methyl Ethyl Ketone	3.20E-05	Dioxins	3.1E-07
Cresols	2.88E-03	Furans	1.4E-06

TABLE C-10

EMISSION ESTIMATES FOR UPSET SCENARIOS

Operations	Emission Rate @ 150 000 t/a (g/s)	Emission Rate @ 300 000 t/a (g/s)
Transportation Spill		
Methylene Chloride	170.0	170.0
Organic Waste Reception		
Methylene Chloride	8.1E-1	8.1E-1
Inorganic Waste Reception		
Hydrogen Cyanide	1.5E-3	1.5E-3
Tank Farm Fire		
Methylene Chloride	1.3E+3	1.3E+3
Cyanide Treatment		
Hydrogen Cyanide	2.9E-4	2.9E-4
Sulfide Treatment		
Hydrogen Sulfide	5.5E-4	5.5E-4
Mixing of Incompatible Wastes		
Hydrogen Sulfide	4.6E-1	4.6E-1
Incinerator Scrubber Failure		
Hydrogen Chloride	69.0	138.0
Sulfur Dioxide	53.5	107.0
Hydrogen Fluoride	1.6	3.1
Nitrogen Oxides	1.6	3.1

Fugitive emissions of inorganics, for which our analysis was confined to hydrogen cyanide, occur for two cases. For inorganic waste reception, the estimated emissions at 150 000 t/a and 300 000 t/a were 1.6×10^{-6}. These emissions were considered to represent a negligible risk relative to the fugitive emissions and were not considered further in the risk assessment.

Incinerator emissions are presented in Table C-9 for a plant capacity of 300 000 t/a, and include the estimated values for organics, metals, acid gases and dioxins/furans. At a reduced plant capacity of 150 000 t/a these values would all be halved.

Emissions under upset conditions are presented in Table C-10 for the following upset or accident conditions: transportation spill; scrubber failure for organic waste reception; scrubber failure for inorganic waste reception; the tank farm fire; pH excursions during cyanide treatment; pH excursions during sulfide treatment; mixing of incompatible wastes; and incinerator scrubber failure. We note that, with the exception of the incinerator scrubber failure, each of these emission rates is an episodic event unaffected by changes in plant capacity. For the incinerator scrubber failure, since the hypothesized failure mode was based on power failure, both incinerator units would malfunction, so that the emission rate at 300 000 t/a would be double that at 150 000 t/a. Finally, of all the failure modes considered, only the transportation spill, tank farm fire, mixing of incompatible wastes and incinerator scrubber failure were considered in the risk assessment. The other emissions were considered to represent a negligible risk relative to other accident or upset conditions identified.

REFERENCES

Abkowitz, M., Eiger, A., Srinivasan, S. 1984. Assessing the Releases and Costs Associated with Truck Transport of Hazardous Wastes. Prepared for the Office of Solid Waste, USEPA and ICF Inc., Draft Final Report, Ch. 4.

American Petroleum Institute (API). 1976. Recommended Practice for the Design and Installation of Pressure-Relieveing Systems in Refineries. Part 1-Design. API Recommended Practice 520. Fourth Edition.

CRC Handbook of Chemistry and Physics, eds. Weast, R.C., and Astle, M.J. CRC Press, Inc., Florida, 1983.

M.M. Dillon Limited. 1984. Site Selection Process. Phase 3B: Selection of Candidates Sites: Facilities Characteristics and Technical Buffer Zones. Prepared for Ontario Waste Management Corporation.

M.M. Dillon Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Transportation. Prepared for Ontario Waste Management Corporation.

General Electric Co. (GE). 1980. Material Safety Data Sheets. Nielsen, J.M. ed.

Lyman, W. J., Rechl, W.F., Rosenblatt, D.H. 1982. Handbook of Chemical Property Estimation Methods. McGraw Hill Book Company.

Mabey, W.R., Smith, J.H., Podoll, R.T., Johnson, H.L., Mill, T., Chou, T.W., Gates, J., Partridge, I.W., Jaber, H., and Vandenberg, O. 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, D.C. EPA Contracts 68-01-3867 and 68-03-2981.

Monenco Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management Corporation.

Ontario Research Foundation (ORF). 1985. Emissions Related Studies: Preliminary Estimates of Air Emisssion. In preparation for Ontario Waste Management Corporation. January.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals. 2nd ed. Van Nostrand Reinhold Co., New York.

APPENDIX D

DERIVATION OF ENVIRONMENTAL TRANSPORT DATA

C O N T E N T S

		Page
D.1	INTRODUCTION	D-1
D.2	ESTIMATION OF ATMOSPHERIC DEPOSITION RATES	D-1
D.3	ESTIMATION OF LEAF SURFACE DEPOSITION FACTOR	D-4
	D.3.1 Model for Leaf Surface Deposition	D-4
	D.3.2 Estimation of Degradation Rate	D-6
D.4	ESTIMATION OF UPTAKE FROM SOIL	D-10
	D.4.1 Model for Plant Uptake	D-10
	D.4.2 Estimation of Loss Terms	D-13
D.5	ESTIMATION OF RETENTION IN CATTLE TISSUES AND MILK	D-20
D.6	ESTIMATION OF CONCENTRATIONS IN CISTERNS AND PONDS	D-27
D.7	SUMMARY OF RESULTS	D-28
REFERENCES		D-33

LIST OF TABLES

		Page
D.1	Deposition Velocities	D-3
D.2	Leafy Vegetable Crop Yield and Consumption	D-7
D.3	Data Inputs For Derivation of the Plant Surface Degradation Rate Constant	D-11
D.4	Soil to Plant Partition Coefficients	D-14
D.5	Data Inputs for Estimation of the Soil Volatilization Rate Constant	D-17
D.6	Distribution Coefficients for Metals	D-19
D.7	Soil Loss Rate Constants	D-21
D.8	F_B and F_m Values for Metals	D-22
D.9	Octanol - Water Partition Coefficients	D-23
D.10	Food Factors for Cattle	D-24
D.11	Concentrations of Chemicals in Leafy Vegetables	D-29
D.12	Concentrations in Cattle Tissues	D-30
D.13	Concentrations in Milk	D-31
D.14	Cistern and Pond Concentrations	D-32

D.1 INTRODUCTION

This appendix presents the methodology and supporting data for the estimation of environmental fate and transport of chemicals released from the OWMC facility. The appendix provides inputs to the estimation of releases from transportation spills and to the estimation of indirect exposures via consumption of crops and livestock products. The appendix is organized in the following sections:

- Estimation of Atmospheric Deposition Rates
- Estimation of Leaf Surface Deposition Factor
- Estimation of Uptake From Soil
- Estimation of Retention in Cattle Tissues and Milk

D.2 ESTIMATION OF ATMOSPHERIC DEPOSITION RATES

Chemicals in the atmosphere can come into contact with soil and plant surfaces through wet and dry deposition processes. Dry deposition describes the contact of very small particles and gases in the atmosphere due to atmospheric turbulence and molecular motion. Upon contact, the particle or gas can be retained due to effects such as absorption.

The dry deposition rate is defined by Hanna et al. (1982) as:

$$d_{dry} = C\ V_d$$

where d_{dry} = dry deposition rate, $(\mu g/m^2)/s$

C = chemical concentration near the surface, $\mu g/m^3$

V_d = deposition velocity, m/s

The deposition velocities for the chemicals of concern are presented in Table D.1, as provided by Ontario Research Foundation. The wet deposition process describes the contact of chemicals in the atmosphere due to washout by precipitation. In the case of gases, equilibrium partitioning into raindrops is the driving force for removal of the chemical from the atmosphere. It has been suggested that equilibrium is reached between gas and water droplets after a fall of approximately ten metres (Slinn et al., 1981). Such chemical partitioning between water and air is described in a "washout ratio." Although this ratio varies depending upon the nature of the chemical and whether it is in the form of a gas, colloid, or particle, conservative values were assumed for metals and organic chemicals.

Wet deposition can be defined by the following equation (Hanna et al., 1982):

$$d_{wet} = C\ P\ \frac{1}{D}\ W$$

TABLE D.1

DEPOSITION VELOCITIES

CHEMICAL CONSTITUENT	DEPOSITION VELOCITY Vd (m/s)
TRICHLOROETHYLENE	0.01
PERCHLOROETHYLENE	0.01
CHLOROFORM	0.01
1,1,1-TRICHLOROETHANE	0.01
HYDRAZINE	0.01
ACRYLONITRILE	0.01
METHYLENE CHLORIDE	0.01
PYRIDINE	0.01
BENZENE	0.01
TOLUENE	0.01
PHENOL	0.01
FORMALDEHYDE	0.01
PETROLEUM DISTILLATES	0.01
TETRAETHYL LEAD	0.01
PCB	0.005
ETHANOL	0.01
STYRENE	0.01
METHYL ETHYL KETONE	0.01
CRESOLS	0.01
DIOXINS/FURANS	0.0025
LEAD	0.003
ZINC	0.0062
NICKEL	0.0045
CHROMIUM (VI)	0.005
ARSENIC	0.0022
CADMIUM	0.0045
MERCURY	0.01
COPPER	0.005
BERYLLIUM	0.01

Source: Ontario Research Foundation

where d_{wet} = wet deposition rate, $(\mu g/m^2)/s$

P = annual precipitation = 0.9423 m (Environment Canada, 1982)

D = density of air = 1.2×10^9 $\mu g/m^3$

W = washout ratio = 500 for metals and 50 for organic chemicals (Ontario Research Foundation)

C = concentration of chemical in the air, $\mu g/m^3$

The overall deposition rate used to determine the concentration of pollutant in the plant was obtained from the sum of dry and wet deposition processes by the following equation:

$$d = C \left[V_d + P \frac{1}{D} W\right]$$

where d = total chemical deposition rate, $(\mu g/m^2)/s$

C = chemical concentration in air, $\mu g/m^3$

V_d = deposition velocity, m/s

P = average annual precipitation, m/s

D = density of air, $\mu g/m^3$

W = washout ratio, unitless

D.3 ESTIMATION OF LEAF SURFACE DEPOSITION FACTOR

D.3.1 MODEL FOR LEAF SURFACE DEPOSITION

Chemicals in the atmosphere can be deposited onto leaf surfaces due to the dry and wet deposition processes described above. Whether this deposition plays a significant role in plant accumulation is not only controlled by the magnitude

of this deposition but also by such factors as the fraction intercepted by vegetation, crop yield and various chemical specific surface degradation processes. Thus, the following equation was used to define the leaf surface deposition factor (Holton et al., 1984):

$$A_d = \frac{r[1 - e^{-kt}]}{Y \quad k}$$

where A_d = leaf surface deposition factor, $m^2.s/kg$

k = total rate constant for plant leaf degradation processes, s^{-1}

t = time of the growing season, s

Y = yield of the plant (wet weight), kg/m^2

r = interception fraction of plant = 0.39

The extent of surface coverage is determined by the interception fraction of the plant. Since literature references were unavailable, the interception fraction for leafy vegetables was based on a theoretical model developed by Baes et al. (1984). The model generates a range of possible interception fractions given various assumptions of planting practices. Baes et al. considered leafy vegetables such as lettuce, cabbage, greens (collards, kale, spinach), broccoli, mint, celery, cauliflower, green onions, escarole, and brussel sprouts in the analysis. The maximum interception fraction for mature leafy vegetables was found to be 0.79. From this theoretical maximum interception, Baes et al. generated a maximum interception fraction over the time in the field by taking into account the logistic growth characteristics

of plants. The theoretical maximum interception fraction was determined to be 0.39. This theoretical maximum was used in this analysis both for leafy vegetables and for grains and grasses consumed by cattle.

For the estimation of indirect exposure, a growing season of five months was assumed. Leafy vegetables were selected for modeling human exposure due to ingestion of locally grown crops because they are considered to accumulate chemicals to a greater degree than non-leafy vegetables. Crop yield was also considered an important factor since high crop yield has the effect of "diluting" the pollutant concentration in the plant by lowering the surface deposition factor. Crop yields were obtained from Ecologistics for the vegetables determined to have the highest potential to bioaccumulate, specifically, asparagus, lettuce, broccoli, cauliflower, and cabbage. It was then conservatively assumed that an individual's dietary consumption of leafy vegetables is made up entirely of these five vegetables, and a weighted average crop yield was developed based on typical consumption levels for each vegetable (see USEPA, 1978). The crop yields and the weighting factors are shown on Table D.2.

D.3.2 ESTIMATION OF DEGRADATION RATE

Chemicals on a leaf surface can undergo degradative processes such as photolysis, can be washed off by precipitation or lost through volatilization. These processes can decrease the amount of chemical deposited on a leaf surface and were expressed in the model as a total rate constant for surface degradation (K).

TABLE D.2

LEAFY VEGETABLE CROP YIELD AND CONSUMPTION

Crop	Yield[1] kg/m^2	Intake[2] lbs/wk/household
Asparagus	0.17	0.14
Broccoli	0.88	0.10
Cabbage	4.09	0.72
Cauliflower	2.09	0.07
Lettuce	2.20	1.28
	Weighted Average = 2.61 kg/m^2	

Source: [1]Ecologistics
[2]USEPA, 1978. Food Factor Tables.

Photolysis

Photolysis describes chemical losses due to transformations caused by the energy of the sun. Organic chemicals are particularly susceptible to such transformations and the photolysis rate constant can be the largest contributor to the total surface loss rate constant, thereby dominating the degradative processes. Of the chemicals selected for modeling, the fate of dioxin could most be affected by photolysis. Dioxin can undergo photolysis under certain conditions (e.g., in the presence of a hydrogen donor). Dioxin formed during incineration, however, would tend to be bound to fly ash. In this state, it is uncertain that photolysis would occur. For these reasons, photolysis was not considered in the analysis for dioxin. Furthermore, photolytic degradation was similarly omitted in an effort to introduce an additional margin of safety in considering the degradation of all organic chemicals selected for modeling.

Weathering

The rate constant for weathering (K_w) describes the loss of a deposited chemical due to wash off by precipitation. A weathering half-life of 15 days was assumed (Willis et al. 1980) for all the chemicals modeled in the risk assessment. This half-life was based on leaf wash-off of the pesticide endrin, a highly hydrophobic chemical which would be expected to adsorb strongly to soil or leaf surfaces. The weathering half-life of 15 days is comparable to the 14 day half-life used by Baes et al. (1984) for the wash-off of metals from a leaf surface. The weathering rate constant is defined by:

$$K_w = \frac{0.693}{t_{1/2}} = 5.34 \times 10^{-7}\ s^{-1}$$

where K_w = weathering rate constant, s^{-1}

$t_{1/2}$ = half-life of chemical on the leaf due to washoff = 15 days

Volatilization

Volatilization of organic chemicals from a leaf surface can represent a significant loss. This loss could occur after dry deposition of the chemical onto the leaf surface, or as leaf surface moisture containing the chemical evaporates. Chemicals would be expected to be found in leaf surface moisture due to partitioning after dry deposition, or due to the washout of wet deposition. Adsorption of chemicals onto the waxy cuticle of a leaf would decrease chemical volatilization losses, thereby providing a more conservative estimate of exposure from surface deposition.

Only organic chemical volatilization was modeled since metals deposited onto leaf surfaces in the form of oxides or chlorides would not be lost through volatilization due to their extremely low vapor pressures. No volatilization was assumed for dioxin due to the fact that dioxin deposited onto a leaf surface would tend to be bound to fly ash, and hence would not be readily lost through volatilization.

An empirically derived soil surface volatilization model developed by Dow Chemical Company was used to estimate volatilization from a leaf surface (Lyman et al., 1982). The model incorporates basic properties such as vapor pressure, water solubility and organic carbon adsorption which are also significant properties in volatilization of chemicals from a leaf surface. Assuming that a leaf surface contains 100% organic carbon, the volatilization rate constant can be estimated by:

$$K_V = 67\,697 \frac{P}{K_{OC}\, S}$$

where K_V = volatilization rate constant, s^{-1}

P = vapor pressure of the chemical, Pa

S = water solubility of the chemical, mg/L

K_{OC} = soil (organic carbon) adsorption coefficient, $(\mu g/g)/(\mu g/ml)$

67 697 = unit conversion factor

A total rate constant for plant leaf degradation processes based on losses due to weathering and volatilization was then derived:

$$K = K_v + K_w$$

where K = total rate constant for plant surface degradation, s^{-1}

K_v = rate constant for volatilization from the leaf surface, s^{-1}

K_w = rate constant for wash off due to precipitation, s^{-1}

As mentioned previously, metals deposited onto leaf surfaces would not be lost through volatilization. Thus, the total rate constant for plant leaf degradation processes for metals equals the rate constant for wash off due to precipitation. The total rate constants for the organic chemicals of concern and the contributions from volatilization are presented in Table D.3.

D.4 ESTIMATION OF UPTAKE FROM SOIL

D.4.1 MODEL FOR PLANT UPTAKE

Chemicals deposited on the soil surface can leach into the soil solution in the root zone and be taken up by a plant. The extent of plant uptake was estimated by a plant-soil partition coefficient, a parameter that describes how a chemical will ideally distribute itself between a plant and soil given a net concentration in the soil. In addition to uptake by the plant, processes such as downward chemical leaching into soil depths, degradation by soil microorganisms, and volatilization upward through pores in the soil also contribute to the removal of a chemical from the root zone. These processes make the chemical less available for plant uptake.

TABLE D.3

DATA INPUTS FOR DERIVATION OF THE
PLANT SURFACE DEGRADATION RATE CONSTANT

CHEMICAL CONSTITUENT	SOLUBILITY AT 25C (mg/l)	VAPOR PRESSURE AT 25C (mm Hg)	Koc	PLANT VOLATIZ. RATE CONSTANT (1/s)	TOTAL PLANT SURFACE DEG. RATE (1/s)
TRICHLOROETHYLENE	1.10E+03	7.38E+01	1.26E+02	2.71E-01	2.71E-01
PERCHLOROETHYLENE	1.50E+02	1.40E+01	3.64E+02	1.31E-01	1.31E-01
CHLOROFORM	9.30E+03	1.93E+02	4.40E+01	2.41E-01	2.41E-01
1,1,1-TRICHLOROETHANE	4.40E+03	1.00E+02	1.52E+02	7.61E-02	7.61E-02
HYDRAZINE	1.00E+06	1.60E+01	4.30E+00	1.89E-03	1.89E-03
ACRYLONITRILE	7.90E+04	1.05E+02	8.50E-01	7.96E-01	7.96E-01
METHYLENE CHLORIDE	1.67E+04	4.33E+02	8.80E+00	1.50E+00	1.50E+00
PYRIDINE	3.00E+08	2.06E+01	2.65E-02	1.32E-03	1.32E-03
BENZENE	1.78E+03	9.45E+01	6.50E+01	4.16E-01	4.16E-01
TOLUENE	5.15E+02	2.82E+01	3.00E+02	9.30E-02	9.30E-02
PHENOL	9.30E+04	5.00E-01	1.42E+01	1.93E-04	1.93E-04
FORMALDEHYDE	6.00E+05	3.83E+03	1.50E-01	2.17E+01	2.17E+01
PETROLEUM DISTILLATES	9.34E-02	4.00E+00	1.50E+04	1.36E+00	1.36E+00
TETRAETHYL LEAD	3.10E+01	1.52E-01	7.30E+03	3.42E-04	3.42E-04
PCB	3.10E-02	7.71E-05	5.30E+05	2.39E-06	2.92E-06
ETHANOL	1.00E+06	5.86E+01	1.60E+01	1.86E-03	1.87E-03
STYRENE	3.00E+02	6.12E+00	1.89E+02	5.49E-02	5.49E-02
METHYL ETHYL KETONE	3.53E+05	1.00E+02	1.00E-02	1.44E+01	1.44E+01
CRESOLS	2.35E+04	1.75E-01	3.54E+01	1.07E-04	1.08E-04
DIOXINS	1.97E-05	1.00E-06	3.30E+06	0.00E+00	5.30E-07
FURANS	1.50E-02	2.00E-06	7.89E+05	0.00E+00	5.30E-07

NOTE: K = kv + Kw where Kw = 5.34E-07

The extent of plant uptake was estimated using the following equation (Holton et al., 1984):

$$Up = \frac{B[1 - e^{-kt}]}{D\ z\ k}$$

where Up = plant uptake factor, $m^2.s/kg$

B = soil-plant partition coefficient, unitless

D = soil density, kg/m^3

z = root zone depth, m

k = total rate constant for soil degradation processes, s^{-1}

t = growing season, s

Soil-plant partition coefficients can be defined as the ratio of the concentration of chemical in the plant to the concentration of the chemical in the soil, obtained under equilibrium or steady state conditions. Such partition coefficients are as yet poorly defined and are not easily extrapolated from a given system due to variations in both soils and plant species. Two sources of information were considered in developing an approach to estimating soil-plant partition coefficients for this risk assessment. The first of these was a food chain risk assessment done by the US Army Medical Bioengineering Research and Development Laboratory (Rosenblatt and Small, 1981) . This work suggests that there may be some correlation between chemical solubility and the soil-plant partition coefficient and used a value for the partition coefficient of 1.0 for substances with solubilities between 0.002 to 10,000 ppm. The solubilities of the chemicals examined in this risk

assessment range from 2×10^{-4} ppm to miscible with water, suggesting that the soil-plant partition coefficient of 1.0 would be applicable for the majority of surrogate chemicals.

The second source of data was the microcosm study of Figge, Klahn and Koch (1983) which derives partition coefficients based on the percentage of radioactivity recovered in plants and soil. This work was utilized because the partitioning of a wide variety of chemicals (specifically, perylene, 2,4,5-T, 4-nitrophenol, DDT, DOP, PCP, $HgCl_2$, phenol, 2-nitrophenol, aniline, trichloroethylene, and toluene) between air, soil, plants and percolating water in a microcosm environment was addressed. The maximum partition coefficient derived from this work was 1.8 in the case of phenol. A default value of 2.0 was therefore adopted for all organic chemicals as a conservative approximation.

Baes et al. (1984) have calculated partition coefficients for radionuclides. The soil to plant partition coefficients calculated by Baes were used in the analysis for metals. These values are shown in Table D.4.

D.4.2 ESTIMATION OF LOSS TERMS

The model developed by Holton et al. (1984) addresses soil loss due to biodegradation and leaching. This analysis extends the loss process to include volatilization upward from the root zone through soil pores by the method of Bomberger et al. (1982). Such losses could be significant in removing chemicals of high volatility which do not tend to adsorb onto soil particles. In the case of dioxin bound to fly ash, no volatilization was assumed.

TABLE D.4

SOIL TO PLANT PARTITION COEFFICIENTS

Chemical Constituent	B
Lead	0.045
Zinc	1.5
Nickel	0.06
Chromium	0.0075
Arsenic	0.04
Cadmium	0.55
Mercury	0.9
Copper	0.4
Beryllium	0.01

Source: Baes et al. (1984)

Volatilization

The derivation of a soil volatilization rate constant was applied only to organic chemicals since inorganic chemicals would not be lost through volatilization. The rate constant was based on a half life for volatilization of chemicals from a root zone depth of 15 cm:

$$K_{SV} = \frac{0.693}{t_{1/2}}$$

where: K_{SV} = volatilization rate constant, s^{-1}

$t_{1/2}$ = half-life for soil volatilization, s

The half-life for volatilization from a depth of 15 cm was calculated by the method of Bomberger et al. (1982):

$$t_{1/2} = \frac{L^2}{4.16\ D^A_{soil}}$$

where $t_{1/2}$ = the half-life for diffusion of a chemical from the soil, s^{-1}

L = depth at which the chemical is found; root zone = 15 cm

D^A_{soil} = diffusion coefficient for the chemical in soil air, cm^2/s

The diffusion coefficient for the chemical in soil air was calculated in the following manner (Bomberger et al., 1982):

$$D^A_{soil} = \frac{D^A T_A}{\alpha}$$

where D^A_{soil} = diffusion coefficient for the chemical in soil air, cm^2/s

D^A = diffusion coefficient for the chemical in free air, cm^2/s

T_A = correction for the porosity of the soil

α = correction factor for chemical adsorption on the soil solid and dissolution in the soil water

The correction factors are defined in the following manner:

$$T_A = \frac{0_A^{10/3}}{(0_A + 0_w)^2}$$

where O_A = volume fraction of the soil that is air = 0.26

O_w = volume fraction of the soil that is water = 0.24

and $$\alpha = O_A + \frac{O_w}{H} + \frac{\rho K_{oc} F_{oc}}{H}$$

where H = Henry's Law constant, dimensionless

ρ = soil bulk density = 1.35 g/ml

K_{oc} = soil adsorption coefficient based on organic carbon

F_{oc} = fraction of organic carbon in the soil = 0.01

O_A = volume fraction of the soil that is air, dimensionless

O_w = volume fraction of the soil that is water, dimensionless

It was assumed that the fraction of organic carbon in the soil would typically be in the order of 1% (Gartner Lee Associates). The Henry's Law constants and diffusion coefficients for the chemicals of concern are presented in Table D.5.

Leaching

Chemicals that are soluble in water will leach from the soil to some degree and tend to migrate vertically downwards from the root zone. The extent of such migration is dependent upon the amount of the precipitation, the soil porosity and the tendency of the chemical to resist movement by adsorption onto organic carbon found in the soil. This tendency is expressed by a partition coefficient (K_{oc}) which describes the distribution of a chemical between soil organic carbon and water. Therefore, soils with a large fraction

TABLE D.5

DATA INPUTS FOR ESTIMATION OF
THE SOIL VOLATILIZATION RATE CONSTANT

CHEMICAL CONSTITUENT	DIFF IN AIR (cm^2/s)	HENRY'S LAW CONSTANT (unitless)
TRICHLOROETHYLENE	8.75E-02	3.79E-01
PERCHLOROETHYLENE	7.97E-02	6.38E-01
CHLOROFORM	8.88E-02	1.20E-01
1,1,1-TRICHLOROETHANE	7.94E-02	1.25E+00
HYDRAZINE	1.90E-01	5.40E-07
ACRYLONITRILE	1.06E-01	3.60E-03
METHYLENE CHLORIDE	1.04E-01	8.46E-02
PYRIDINE	9.50E-02	2.90E-07
BENZENE	9.32E-02	2.25E-01
TOLUENE	8.49E-02	2.78E-01
PHENOL	8.50E-02	1.86E-05
FORMALDEHYDE	1.70E-01	1.15E-02
PETROLEUM DISTILLATES	7.57E-02	2.38E+02
TETRAETHYL LEAD	1.00E-02	3.28E+00
PCB	4.00E-02	1.07E-01
ETHANOL	1.20E-01	1.46E-04
STYRENE	7.50E-02	1.87E-01
METHYL ETHYL KETONE	8.94E-02	1.50E-03
CRESOLS	8.10E-02	4.34E-05
DIOXINS	5.00E-02	8.58E-02
FURANS	5.06E-02	2.51E-03

of organic carbon will have a greater tendency to retain certain organic chemicals than soils with a low fraction of organic carbon. As in the calculation of volatilization, it was assumed that the soils in question contain 1% organic carbon (Gartner Lee Associates) in order to calculate a leaching loss rate constant.

The leaching loss rate constant was calculated by the method of Baes et al. (1984) for a clay soil of 1% organic carbon and a volumetric water content of 0.24:

$$k_1 = \frac{P - E}{O_w \, z \, [1 + (\rho K_d)]}$$
$$O_w$$

where k_1 = rate constant for leaching loss, s^{-1}

P = annual average total precipitation = 2.991×10^{-6} cm/s (Environment Canada, 1982)

E = annual average evapotranspiration = 1.693×10^{-6} cm/s (Environment Canada, 1982)

O_w = volumetric water content of the soil = 0.24

z = depth of soil layer from which leaching occurs = 15 cm

ρ = soil bulk density = 1.35 g ml^{-1}

K_d = distribution coefficient (ml/g)

For organic chemicals, the distribution coefficient was defined by:

$$K_d = F_{oc} K_{oc}$$

where K_{oc} = soil adsorption coefficient

F_{oc} = fraction of organic carbon in the soil = 0.01

For the inorganic compounds, K_d values derived by Baes et al. were adopted and are shown in Table D.6.

TABLE D.6

DISTRIBUTION COEFFICIENTS FOR METALS

Chemical Constituent	K_d
Lead	900
Zinc	40
Nickel	150
Chromium	850
Arsenic	200
Cadmium	65
Mercury	10
Copper	35
Beryllium	650

Source: Baes et. al. (1984)

Biodegradation

Biodegradation in soils is highly variable. Rate constants were obtained from the literature and where a range of values was indicated, the most conservative number (lowest rate of biodegradation) was selected. In the case of metals and dioxin bound to fly ash, no biodegradation was assumed. The soil volatilization rate constant, the leaching loss rate constant,

biodegradation rate constant and the overall soil loss rate constant are shown on Table D.7.

D.5 ESTIMATION OF RETENTION IN CATTLE TISSUES AND MILK

The concentration of chemical found in beef and milk due to leaf surface deposition and plant uptake was estimated by the model of Holton et al. (1984). The model requires the use of cattle dietary assumptions, the concentration of the chemical found in cattle food crops and the fraction of the chemical that is retained in beef and milk from the consumption of exposed food crops:

$$C_B = F_B (Q_B C_g)$$

$$C_m = F_m (Q_m C_g)$$

where C_B = estimated concentration of the contaminant in beef, ug/kg

C_m = estimated concentration of the contaminant in milk, ug/kg

F_B = fraction of the contaminant consumed each day which is transported and retained in beef, d/kg

F_m = fraction of the contaminant consumed each day which is transported and retained in milk, d/kg

Q_B = quantity of non-leafy vegetation (hay, mixed grains, corn silage eaten by beef cattle each day (wet weight), kg/d (Ecologistics)

Q_m = quantity of non-leafy vegetation (hay, mixed grains, corn silage) eaten by milk cows each day (wet weight), kg/d (Ecologistics)

TABLE D.7

SOIL LOSS RATE CONSTANTS

CHEMICAL CONSTITUENT	SOIL VOLATIZ. RATE CONSTANT (1/s)	SOIL LEACHING RATE CONSTANT (1/s)	BIODEG. RATE CONSTANT (1/s)	TOTAL SOIL LOSS RATE CONSTANT (1/s)
TRICHLOROETHYLENE	9.35E-06	4.47E-08	2.80E-11	9.39E-06
PERCHLOROETHYLENE	5.50E-06	1.68E-08	2.80E-11	5.51E-06
CHLOROFORM	7.08E-06	1.04E-07	1.10E-06	8.29E-06
1.1.1-TRICHLOROETHANE	2.18E-05	3.78E-08	4.40E-07	2.23E-05
HYDRAZINE	1.98E-10	2.90E-07	8.30E-07	1.12E-06
ACRYLONITRILE	8.69E-07	3.44E-07	8.30E-10	1.21E-06
METHYLENE CHLORIDE	1.33E-05	2.41E-07	2.30E-06	1.58E-05
PYRIDINE	6.59E-11	3.59E-07	4.50E-05	4.54E-05
BENZENE	1.03E-05	7.76E-08	5.00E-07	1.08E-05
TOLUENE	3.11E-06	2.02E-08	2.80E-08	3.16E-06
PHENOL	2.11E-09	2.01E-07	8.30E-07	1.03E-06
FORMALDEHYDE	4.59E-06	3.57E-07	3.30E-05	3.79E-05
PETROLEUM DISTILLATES	3.72E-05	4.01E-10	1.20E-06	3.84E-05
TETRAETHYL LEAD	1.89E-07	8.78E-10	0.00E+00	1.90E-07
PCB	3.44E-10	1.21E-11	8.30E-10	1.19E-09
ETHANOL	2.21E-08	1.90E-07	3.30E-05	3.32E-05
STYRENE	2.84E-06	2.11E-08	8.30E-10	2.87E-06
METHYL ETHYL KETONE	3.21E-07	3.60E-07	6.10E-06	6.78E-06
CRESOLS	2.82E-09	1.21E-07	2.27E-05	2.28E-05
DIOXINS/FURANS	0.00E+00	1.95E-12	0.00E+00	1.95E-12
	0.00E+00	8.15E-12	0.00E+00	8.15E-12
LEAD		7.12E-11		7.12E-11
ZINC		1.60E-09		1.60E-09
NICKEL		4.27E-10		4.27E-10
CHROMIUM (VI)		7.54E-11		7.54E-11
ARSENIC		3.20E-10		3.20E-10
CADMIUM		9.80E-10		9.80E-09
MERCURY		6.30E-09		6.30E-09
COPPER		1.82E-09		1.82E-09
BERYLLIUM		9.86E-11		9.86E-11

C_g = estimated concentration of contaminant in grains and grasses, ug/kg

The fraction of the chemical that is retained in the beef and milk after the consumption of exposed grain, hay and corn silage by cattle (F_B and F_m) were derived by Baes et al. (1984) for metals. These values were adopted and are shown in Table D.8. For organic chemicals, F_B and F_m were estimated using correlation equations developed by Holton et al. (1984) and based on the work of Kenaga. These equations use the octanol-water partition coefficient (K_{ow}) which describes how a chemical will distribute itself between a water phase and a hydrophobic octanol phase. Octanol partitioning has been used as an estimate of the lipid solubility of a chemical, and hence how it will be accumulated in body fat. Octanol-water partition coefficients for the chemicals of concern are shown in Table D.9.

TABLE D.8

F_B AND F_m VALUES FOR METALS

Chemical Constituent	F_B	F_m
Lead	3×10^{-4}	2.5×10^{-4}
Zinc	1×10^{-1}	1×10^{-2}
Nickel	6×10^{-3}	1×10^{-3}
Chromium	5.5×10^{-3}	1.5×10^{-3}
Arsenic	2×10^{-3}	6×10^{-5}
Cadmium	5.5×10^{-4}	1×10^{-3}
Mercury	2.5×10^{-1}	4.5×10^{-4}
Copper	1×10^{-2}	1.5×10^{-3}
Beryllium	1×10^{-3}	9×10^{-7}

Source: Baes et al. (1984)

TABLE D.9

OCTANOL-WATER PARTITION COEFFICIENTS

CHEMICAL CONSTITUENT	LOG Kow
TRICHLOROETHYLENE	2.42
PERCHLOROETHYLENE	2.6
CHLOROFORM	1.97
1,1,1-TRICHLOROETHANE	2.51
HYDRAZINE	-1.37
ACRYLONITRILE	-0.92
METHYLENE CHLORIDE	1.26
PYRIDINE	0.64
BENZENE	2.13
TOLUENE	2.69
PHENOL	1.46
FORMALDEHYDE	-0.65
PETROLEUM DISTILLATES	0.67
TETRAETHYL LEAD	4.57
PCB	6.04
ETHANOL	-0.32
STYRENE	2.95
METHYL ETHYL KETONE	0.26
CRESOLS	1.98
DIOXINS/FURANS	6.84

In order to estimate F_B and F_m for organic chemicals, the following regression equations were used (Holton et al., 1984):

$$\log F_B = -5.15 + 0.50 (\log K_{ow})$$

$$\log F_m = -6.13 + 0.50 (\log K_{ow})$$

where F_B = fraction of the chemical consumed each day which is transported and retained in beef, d/kg

F_m = fraction of the chemical consumed each day which is transported and retained in milk, d/kg

K_{ow} = octanol/water partition coefficient

The quantity of hay, corn silage and mixed grain consumed by dairy cows and steers per day (Q_g) was provided by Ecologistics and is shown in Table D.10.

TABLE D.10

FOOD FACTORS FOR CATTLE

	Yield kg/m^2	Daily Intake Dairy Cow, kg (wet)	Daily Intake Steer kg (wet)
Hay	0.74	6.8	1.3
Corn silage	3.05	17.1	3.2
Mixed grain	0.30	5.7	7.7
Total		29.6	12.2
		Weighted average = 1.99 kg/m^2	Weighted average = 1.07 kg/m^2

Source: Ecologistics

The concentration of the chemical in the cattle food crops (C_g) was modeled in the same manner as for leafy vegetables:

$$C_g = d[Ad + Up]$$

where Ad = leaf surface deposition factor, m^2.s/kg

Up = soil uptake factor, m^2.s/kg

d = chemical deposition rate, $(ug/m^2)/s$

The leaf deposition factor is defined by Holton et al. (1984) as:

$$Ad = \frac{r\,[1 - e^{-kt}]}{Y\,k}$$

where Ad = leaf surface deposition factor, m^2-s/kg

k = total rate constant for plant leaf degradation processes, s^{-1}

t = time of the growing season, s

Y = yield of the plant (wet weight), kg/m^2

r = interception fraction of the plant, unitless

As for leafy vegetables, a weighted average for crop yield was determined based on consumption of grains and grasses by steers and dairy cows. Due to differences in grain and grass consumption, separate crop yield factors were calculated for dairy cows (1.99 kg/m^2) and for steers (1.07 kg/m^2).

The soil uptake factor is defined by Holton et al. (1984) as:

$$Up = \frac{B\,[1 - e^{-kt}]}{Dzk}$$

where Up = plant uptake factor, $m^2 s/kg$

B = soil-plant partition coefficient, unitless

D = soil density, kg/m^3

z = root zone depth, m

k = total rate constant for soil degradation processes, s^{-1}

t = growing season, s

In the use of soil-plant partition coefficients (B) for grains and grasses, a factor considered was the distribution of chemicals in the plant. Studies by Baes et al. (1984) on radionuclides suggest that only a small fraction of the chemical taken up in the leafy part of the plant may be incorporated into the reproductive part of the plant, i.e., grain. Thus, for grains, Baes suggests that the soil-plant partition coefficient for metals in grains could be one tenth of that predicted for leafy portions of the plant.

It is unclear, however, whether this relationship can reasonably be applied to organic chemicals. Given this uncertainty and to be consistent with the conservative assumptions used throughout the risk assessment, no allowance for reduced uptake by grains was made in estimating chemical concentrations in beef and milk, either for metals or organic chemicals.

D.6 ESTIMATION OF CONCENTRATIONS IN PONDS AND CISTERNS

Chemical concentrations in ponds and cistern water were conceptualized as being controlled by two different processes depending upon whether gaseous or particulate emissions were being considered. For gaseous pollutants, Henry's Law describes how a chemical will distribute itself between air and water under ideal conditions (Lyman et al., 1982):

$$C_A = (C_s)(H)$$

where C_A = equilibrium air concentration of the chemical above the dilute aqueous solution, g/cm^3

C_s = equilibrium aqueous concentration (dilute) of the chemical, g/cm^3

H = Henry's Law constant, dimensionless

For chemicals emitted in particulate form or associated with particulates, i.e., incinerator fly ash, Henry's Law cannot be used to estimate the concentrations in pond and cistern water. For example, metals emitted from incinerators will not remain as gas, but will quickly adsorb to fly ash or exist as solid oxides. For particulate emissions, therefore, concentrations were estimated by the mass of material deposited into a volume of water, and subsequently released from the particles by desorption.

Ponds in the vicinity of the candidate sites are typically 0.6 m to 7.6 m in depth (Gartner Lee Associates). Neglecting any dilution afforded by the initial contents of the pond, the long term concentration in such a pond can be conservatively estimated as:

$$C_m = \frac{(d)(k_s)}{P-E}$$

where C_m = concentration of metal in the pond, g/m^3

d = deposition rate, $g/m^2.a$

P = average annual precipitation, m/a

E = average annual evapotranspiration, m/a

k_s = sediment water partition coefficient

The particulate deposition approach was used in estimating chemical concentrations for all metals emitted from the incinerator. As a conservative assumption, it was also assumed that the sediment-water partition coefficient, i.e., the ratio of the mass of chemical in solution to the mass of chemical adsorbed on the sediment, would be one. For organic chemicals, the Henry's Law approach was applied with the exception of dioxins. Since dioxins may be strongly adsorbed to fly ash emitted from the incinerator, both the Henry's Law and particulate deposition approaches were considered. A sediment-soil partition coefficient of 0.09 was adopted as the lowest value found in reviewing the literature (Matsumura and Ward, 1976). Using both methods the particulate deposition approach was found to result in the highest water concentrations. Consistent with the conservative approach used throughout the generic risk assessment, this higher value was adopted for assessing exposure.

D.7 SUMMARY OF RESULTS

The chemical concentrations predicted in leafy vegetables, cattle tissues and milk, and ponds and cisterns are presented in Tables D.11, D.12, D.13 and D.14, respectively.

TABLE D. 11

CONCENTRATIONS OF CHEMICALS IN LEAFY VEGETABLES

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	3. 77E-01	1. 32E+00	7. 40E-04	4. 44E-03
PERCHLOROETHYLENE	0. 00E+00	0. 00E+00	1. 31E-03	7. 88E-03
CHLOROFORM	4. 40E-01	1. 53E+00	0. 00E+00	0. 00E+00
1, 1, 1-TRICHLOROETHANE	3. 84E-02	1. 31E-01	5. 75E-04	3. 45E-03
HYDRAZINE	0. 00E+00	0. 00E+00	2. 12E-04	1. 27E-03
ACRYLONITRILE	2. 28E-01	8. 00E-01	1. 95E-04	1. 17E-03
METHYLENE CHLORIDE	2. 32E-01	8. 08E-01	4. 48E-06	2. 69E-05
PYRIDINE	5. 28E-03	1. 88E-02	5. 35E-05	3. 21E-04
BENZENE	4. 43E-02	1. 54E-01	1. 31E-04	7. 86E-04
TOLUENE	2. 06E-01	7. 30E-01	2. 15E-03	1. 29E-02
PHENOL	3. 06E-04	1. 12E-03	3. 47E-03	2. 08E-02
FORMALDEHYDE	3. 79E-01	1. 34E+00	2. 41E-05	1. 44E-04
PETROLEUM DISTILLATES	5. 28E-02	1. 92E-01	1. 10E-03	6. 62E-03
TETRAETHYL LEAD	1. 60E-05	5. 81E-05	5. 70E-06	3. 42E-05
PCB	8. 95E-06	3. 29E-05	6. 68E-03	4. 01E-02
ETHANOL	3. 25E-01	1. 16E+00	1. 31E-03	7. 86E-03
STYRENE	3. 89E-01	1. 39E+00	3. 45E-03	2. 07E-02
METHYL ETHYL KETONE	1. 15E-02	4. 02E-02	1. 74E-05	1. 04E-04
CRESOLS	0. 00E+00	0. 00E+00	1. 10E-03	6. 59E-03
LEAD	0. 00E+00	0. 00E+00	8. 22E-04	6. 85E-03
ZINC	0. 00E+00	0. 00E+00	1. 70E-03	1. 41E-02
NICKEL	0. 00E+00	0. 00E+00	7. 80E-06	6. 50E-05
CHROMIUM (VI)	0. 00E+00	0. 00E+00	8. 76E-05	7. 30E-04
ARSENIC	0. 00E+00	0. 00E+00	7. 00E-06	5. 83E-05
CADMIUM	0. 00E+00	0. 00E+00	9. 79E-05	8. 16E-04
MERCURY	0. 00E+00	0. 00E+00	1. 13E-04	9. 41E-04
COPPER	0. 00E+00	0. 00E+00	2. 03E-04	1. 69E-03
BERYLLIUM	0. 00E+00	0. 00E+00	6. 20E-08	5. 17E-07
DIOXINS	0. 00E+00	0. 00E+00	1. 06E-05	6. 38E-05
FURANS	0. 00E+00	0. 00E+00	4. 92E-05	2. 95E-04

TABLE D.12

CONCENTRATIONS IN CATTLE TISSUES

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	5.24E-04	1.83E-03	1.03E-06	6.18E-06
PERCHLOROETHYLENE	0.00E+00	0.00E+00	2.25E-06	1.35E-05
CHLOROFORM	3.64E-04	1.27E-03	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	5.94E-05	2.03E-04	8.89E-07	5.33E-06
HYDRAZINE	0.00E+00	0.00E+00	3.78E-09	2.27E-08
ACRYLONITRILE	6.76E-06	2.38E-05	5.80E-09	3.48E-08
METHYLENE CHLORIDE	8.47E-05	2.95E-04	1.64E-09	9.84E-09
PYRIDINE	1.19E-06	4.25E-06	1.21E-08	7.25E-08
BENZENE	4.41E-05	1.54E-04	1.30E-07	7.83E-07
TOLUENE	3.91E-04	1.38E-03	4.08E-06	2.45E-05
PHENOL	1.48E-07	5.40E-07	1.67E-06	1.00E-05
FORMALDEHYDE	1.54E-05	5.43E-05	9.76E-10	5.86E-09
PETROLEUM DISTILLATES	9.79E-06	3.56E-05	2.05E-07	1.23E-06
TETRAETHYL LEAD	2.65E-07	9.65E-07	9.47E-08	5.68E-07
PCB	9.69E-07	3.56E-06	7.24E-04	4.34E-03
ETHANOL	2.21E-05	7.86E-05	8.91E-08	5.35E-07
STYRENE	9.95E-04	3.56E-03	8.83E-06	5.30E-05
METHYL ETHYL KETONE	1.33E-06	4.64E-06	2.01E-09	1.21E-08
CRESOLS	0.00E+00	0.00E+00	1.68E-06	1.01E-05
LEAD	0.00E+00	0.00E+00	5.16E-03	4.30E-02
ZINC	0.00E+00	0.00E+00	2.66E+00	2.22E+01
NICKEL	0.00E+00	0.00E+00	9.75E-04	8.12E-03
CHROMIUM (VI)	0.00E+00	0.00E+00	1.02E-02	8.50E-02
ARSENIC	0.00E+00	0.00E+00	2.93E-04	2.44E-03
CADMIUM	0.00E+00	0.00E+00	9.89E-04	8.25E-03
MERCURY	0.00E+00	0.00E+00	4.87E-01	4.06E+00
COPPER	0.00E+00	0.00E+00	3.86E-02	3.21E-01
BERYLLIUM	0.00E+00	0.00E+00	1.31E-06	1.09E-05
DIOXINS	0.00E+00	0.00E+00	4.05E-06	2.43E-05
FURANS	0.00E+00	0.00E+00	1.01E-05	6.04E-05

TABLE D.13

CONCENTRATIONS IN MILK

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (ug/kg)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (ug/kg)
TRICHLOROETHYLENE	1.34E-04	4.69E-04	2.64E-07	1.58E-06
PERCHLOROETHYLENE	0.00E+00	0.00E+00	5.75E-07	3.45E-06
CHLOROFORM	9.32E-05	3.24E-04	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	1.52E-05	5.18E-05	2.27E-07	1.36E-06
HYDRAZINE	0.00E+00	0.00E+00	9.62E-10	5.77E-09
ACRYLONITRILE	1.73E-06	6.09E-06	1.48E-09	8.90E-09
METHYLENE CHLORIDE	2.17E-05	7.56E-05	4.20E-10	2.52E-09
PYRIDINE	2.55E-07	9.10E-07	2.59E-09	1.55E-08
BENZENE	1.13E-05	3.93E-05	3.34E-08	2.00E-07
TOLUENE	1.00E-04	3.54E-04	1.04E-06	6.27E-06
PHENOL	3.65E-08	1.33E-07	4.13E-07	2.48E-06
FORMALDEHYDE	3.94E-06	1.39E-05	2.50E-10	1.50E-09
PETROLEUM DISTILLATES	2.51E-06	9.10E-06	5.24E-08	3.14E-07
TETRAETHYL LEAD	6.75E-08	2.46E-07	2.41E-08	1.45E-07
PCB	2.14E-07	7.87E-07	1.60E-04	9.62E-04
ETHANOL	5.08E-06	1.81E-05	2.05E-08	1.23E-07
STYRENE	2.55E-04	9.12E-04	2.26E-06	1.36E-05
METHYL ETHYL KETONE	3.39E-07	1.19E-06	5.15E-10	3.09E-09
CRESOLS	0.00E+00	0.00E+00	2.76E-07	1.66E-06
LEAD	0.00E+00	0.00E+00	5.66E-03	4.72E-02
ZINC	0.00E+00	0.00E+00	4.22E-01	3.52E+00
NICKEL	0.00E+00	0.00E+00	2.14E-04	1.79E-03
CHROMIUM (VI)	0.00E+00	0.00E+00	3.64E-03	3.03E-02
ARSENIC	0.00E+00	0.00E+00	1.16E-05	9.64E-05
CADMIUM	0.00E+00	0.00E+00	2.57E-03	2.14E-02
MERCURY	0.00E+00	0.00E+00	1.30E-03	1.09E-02
COPPER	0.00E+00	0.00E+00	8.08E-03	6.73E-02
BERYLLIUM	0.00E+00	0.00E+00	1.54E-09	1.29E-08
DIOXINS	0.00E+00	0.00E+00	7.01E-07	4.21E-06
FURANS	0.00E+00	0.00E+00	1.74E-06	1.05E-05

TABLE D.14

CISTERN AND POND CONCENTRATIONS

CHEMICAL CONSTITUENT	MAXIMUM ANNUAL CONCENTRATION FROM FUGITIVE EMISSIONS (g/m^3)	MAX TWO-WEEK CONCENTRATION FROM FUGITIVE EMISSIONS (g/m^3)	MAXIMUM ANNUAL CONCENTRATION FROM INCINERATOR EMISSIONS (g/m^3)	MAX TWO-WEEK CONCENTRATION FROM INCINERATOR EMISSIONS (g/m^3)
TRICHLOROETHYLENE	9.11E-08	4.43E-07	1.79E-10	1.49E-09
PERCHLOROETHYLENE	0.00E+00	0.00E+00	1.11E-10	9.22E-10
CHLOROFORM	2.96E-07	1.43E-06	0.00E+00	0.00E+00
1,1,1-TRICHLOROETHANE	6.67E-09	3.16E-08	9.98E-11	8.32E-10
HYDRAZINE	0.00E+00	0.00E+00	4.28E-06	3.56E-05
ACRYLONITRILE	7.49E-07	3.65E-06	6.42E-10	5.35E-09
METHYLENE CHLORIDE	4.23E-07	2.05E-06	8.18E-12	6.82E-11
PYRIDINE	6.59E-03	3.27E-02	6.69E-05	5.57E-04
BENZENE	2.08E-08	1.01E-07	6.15E-11	5.13E-10
TOLUENE	2.28E-08	1.12E-07	2.38E-10	1.99E-09
PHENOL	1.61E-07	8.15E-07	1.82E-06	1.51E-05
FORMALDEHYDE	1.22E-05	5.99E-05	7.75E-10	6.46E-09
PETROLEUM DISTILLATES	8.32E-11	4.20E-10	1.74E-12	1.45E-11
TETRAETHYL LEAD	9.82E-15	4.97E-14	3.51E-15	2.93E-14
PCB	9.26E-14	4.73E-13	6.92E-11	5.77E-10
ETHANOL	6.47E-04	3.20E-03	2.61E-06	2.18E-05
STYRENE	5.82E-08	2.90E-07	5.17E-10	4.31E-09
METHYL ETHYL KETONE	5.06E-07	2.46E-06	7.68E-10	6.40E-09
CRESOLS	0.00E+00	0.00E+00	2.38E-06	1.99E-05
LEAD	0.00E+00	0.00E+00	3.75E-04	3.13E-03
ZINC	0.00E+00	0.00E+00	4.40E-04	3.67E-03
NICKEL	0.00E+00	0.00E+00	3.53E-06	2.94E-05
CHROMIUM (VI)	0.00E+00	0.00E+00	4.08E-05	3.40E-04
ARSENIC	0.00E+00	0.00E+00	3.20E-06	2.66E-05
CADMIUM	0.00E+00	0.00E+00	3.53E-05	2.94E-04
MERCURY	0.00E+00	0.00E+00	3.60E-05	3.00E-04
COPPER	0.00E+00	0.00E+00	7.80E-05	6.50E-04
BERYLLIUM	0.00E+00	0.00E+00	2.88E-08	2.40E-07
DIOXINS	0.00E+00	0.00E+00	2.14E-10	1.78E-09
FURANS	0.00E+00	0.00E+00	9.90E-10	8.25E-09

REFERENCES

Baes, G.F., Sharp, R.D., Sjoreen, A.L. and Shaw, R.W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides in Agriculture. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL-5786).

Bomberger, D.C., Gwinn, J.L., Mabey, W.R., Tuse, D., and Tsong Wen Chou. 1982. Environmental Fate and Transport at the Terrestrial-Atmospheric Interface. Symposium on Models for Predicting Fate of Chemicals in the Environment. American Chemical Society, Division of Pesticides Chemistry, 184th National Meeting, Kansas City, MO.

Environment Canada, 1982. Canadian Climate Normals. Volume 3 - Precipitation: 1951 - 1980.

Hanna, S.R., Briggs, G.A., and Hosker, R.P. 1982. Handbook on Atmospheric Diffusion. National Turbulence and Diffusion Laboratory, National Oceanic and Atmospheric Administration. DOE/TIC-11223.

Holton, G.A., Travis, C.C., Etnier, E.L., O'Donnell, F.R., Hetrick, D.M. and Dixon, E. 1984. Multiple-Pathways Screening-Level Assessment of a Hazardous Waste Incineration Facility. Oak Ridge National Laboratory, Oak Ridge, TN (ORNL/TM-8652).

Lyman, W.J., Reehl, W.F. and Rosenblatt, D.H. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York.

Mackay D., and Matsugu, R.S. 1973. Evaporation Rates of Liquid Hydrocarbon Spills on Land and Water. Canadian Journal of Chemical Engineering. 51:434, August.

Matsumura and Ward. 1976. As cited in Dioxins. Esposito, M.P., Tiernan, T.D. and Dryden, F.E. November 1980.

Monenco Limited. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management Corporation.

Rosenblatt, D.H. and Small, M.J. 1981. Preliminary Pollutant Limit Values for Alabama Army Munition Plant. U.S. Army Medical Bioengineering Research and Development Laboratory.

Shen, T.T. 1982. A Simplified Method of Hazardous Emissions from Waste Lagoons. Presented at the 75th Annual Meeting of the Air Pollution Control Association, New Orleans, June 20-25.

Slinn, S.A. and Slinn, W.G.N. 1981. Modeling of Atmospheric Particulate Deposition to Natural Waters. As cited in Environmental Exposure from Chemicals - Volume I. CRC Press.

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/ February 1982. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

Willis, G.H., Spencer, W.S. and McDowell, L.L. 1980. The Interception of Applied Pesticides by Foliage and their Persistence and Washoff Potential. A Field Scale Model for Chemical Runoff and Erosion from Agricultural Management Systems. USDA Conservation Research Report No. 26.

United States Environmental Protection Agency. 1978. Update of Food Factor Tables. Memo from R. D. Schmitt, Ph. D., Toxicology Branch to Acting Chief, Toxicology Branch. May 1, 1978.

APPENDIX E

RELATIVE RISKS FOR TRANSPORTATION OF SPECIAL WASTES, REAGENTS AND ADDITIVES, AND INTER-SITE TRANSFERS

C O N T E N T S

		Page
E.1	INTRODUCTION	E-1
E.2	METHODOLOGY	E-1
	Wastes	E-4
	Reagents and Additives	E-7
	Inter-Site Transfers	E-11
E.3	RESULTS AND ANALYSIS	E-12
REFERENCES		E-14

LIST OF TABLES

		Page
E-1	Special Wastes	E-5
E-2	Reagants and Additives	E-8
E-3	Inter-Site Transfers	E-12

E.1 INTRODUCTION

Direct exposure to toxic pollutants via the air pathway can occur as a result of a spill on the local access route if a spill occurs in proximity to a neighboring residence. Such spills can occur in the transportation of three groups of commodities associated with the OWMC facility: 1) special wastes transported to the facility; 2) reagents and additives transported to the facility; and 3) treatment residues transferred between units in the event that the components of the facility are located at more than one site. In order to determine the relative risks associated with the transportation of these three commodities, a comparative risk assessment was performed. The risk assessment considered a spill event on the local or regional access route resulting in the release of the material and subsequent air dispersion leading to human exposure by inhalation.

E.2 METHODOLOGY

The comparative risk assessment considered both the consequence or severity of the commodity being spilled and the potential frequency of that event occurring. The severity index was based on the inherent hazard of the

commodity group being transported and the potential for a toxic pollutant to be released to the atmosphere, in either gaseous or particulate form. The severity index was defined for each commodity in the group as:

$$\text{Severity Index} = \text{Acute Toxicity} \times \text{Release Potential}$$

where Acute Toxicity = LD_{50}

Release Potential = Emission rate

Acute toxicity represents the potential for acute health effects on a human receptor. As a relative measure of the severity of such effects, acute toxicity was evaluated using LD_{50} values from animal studies (e.g., the lethal dose of a chemical needed to kill half the exposed population for the most sensitive mammalian species on which it has been tested). When LD_{50} values were unavailable, acute toxicity was derived from the Threshold Limit Value (TLV).

Since each of the three commodity groups contained several commodities of different hazards, e.g., the inter-site transfers includes incinerator ash and oily wastes, a quantity-weighted average for each of the commodity groups was calculated as:

$$\text{Overall Severity Index} = \frac{\sum (SI_i)(Q_i)}{\sum Q_i}$$

where SI_i = severity index for each commodity i

Q_i = quantity of that commodity

In addition to the severity index, the frequency of a spill of the commodity group being transported was assessed based on the quantity of the commodity being transported. It was assumed that commodity groups transported in large quantities would have a greater potential for a spill occurring. Information on the commodities was based on the data base developed by Canviro (1985) to estimate the types and quantities of special wastes generated in Ontario and on the Phase 4A Engineering Report (Monenco, 1985). Estimates are based on the 300 000 t/a ultimate plant throughout.

In employing this methodology, several assumptions were made about the configuration of the spill and the environmental conditions prevailing at the time of the spill, notably wind speed and ambient temperature. In general, average climatic conditions were used for estimating the release potential of volatile and particulate commodities rather than worst case meteorology for comparison of the risk pathways. These assumptions are summarized below:

Spill Area -- The emission rate is directly proportional to the area of the spill. The spill area is assumed to remain constant over the period of concern and is defined by a circle of diameter equal to the width of the paved road surface. Based on information provided by M.M. Dillon Limited, a pavement width of 7.0 m was selected to define the spill area. In the case of solid materials, the spill was assumed to take the shape of a cone with a diameter of 7.0 m and a height of 1.5 m.

Ambient Temperature -- Seasonal temperature differences will affect the emission rate for volatile organics, since the vapor pressure of a chemical increases with increasing temperature. Spills occurring in the summer months will thus result in a higher emission rate than spills in the winter. A temperature of 10^0C was selected for this evaluation as being a typical average annual temperature for Southern Ontario.

Wind Speed -- The emission rate will be affected by changes in wind speed, i.e., the greater the wind speed the greater the emission rate. In the spill scenario considered here, a wind speed of 3 m/s was selected, corresponding to atmospheric stability class D. Stability class D describes a neutral condition and is the most common situation encountered.

The methodology for derivation of the severity index is discussed separately for wastes, reagents and inter-site transfers in the following sections.

WASTES

Since the waste streams accepted at the OWMC facility may contain many hazardous constituents, surrogate chemicals were chosen that represent the most hazardous waste streams to be transported to the facility. The most hazardous waste streams were then used to represent the waste classes. Attention here was focused on those waste classes which were considered most likely to contain significant concentrations of volatile organics. The total quantity of waste containing volatile organics is 59 410 t/a. The remaining 240 590 t/a projected for the 300 000 t/a facility would contain primarily inorganic constituents. The surrogate selection process is addressed in detail in Appendix B of this report. The surrogate chemicals and the quantities of the waste classes they represent are summarized in Table E-1. The acute toxicity was evaluated using LD_{50} values determined for the most sensitive animal species tested.

The release potential was defined as the emission rate of the surrogate chemical from the spill. For volatile organic chemicals, the emission rate was estimated using the following equation for a pure component (Stiver and Mackay, 1982):

$$Q_i = \frac{(Kg)(A)(P_i)(M_i)}{(R)(T)}$$

TABLE E-1

SPECIAL WASTES

	Surrogate Chemical	UCD Category	Waste Quantity (t/a)
Class 1:	Organic Sludges and Still Bottoms		
	Methylene chloride	247	600
	Petroleum distillates	248	2400
	Phenol	257	2000
	Styrene	261	6000
	Acrylonitrile	263	4000
	Trichloroethylene	446	2000
	Formaldehyde	523	2000
Class 2:	Solvents and Organic Solutions		
	Methylene chloride	211	1400
	Trichloroethylene	212	200
	Methylene chloride	213	1000
	Petroleum distillates	214	1200
	Toluene	215	600
	Toluene	216	600
	Ethanol	217	2000
	Acrylonitrile	224	400
Class 3:	Oils and Greases		
	Petroleum distillates	281	2000
Class 4:	Oil and Water Mixtures		
	Trichloroethylene and Benzene	283	8000
Class 5:	Organic and Oily Residuals		
	Petroleum distillates and ethanol	264	20
	Phenol	286	600
	Petroleum distillates	289	6000
	Tetraethyl lead sludge	447	10
Class 7:	Miscellaneous Chemicals and Products		
	Petroleum distillates and ethanol	243	400
	Formaldehyde	245	1000
	Ethanol	416	400
	Pyridine	513	400
Class 8:	Paint and Organic Residuals		
	Methylene chloride	254	1000
	Formaldehyde	262	1000
	Petroleum distillates	454	4000
	Petroleum distillates	455	1000

TABLE E-1 (Continued)

SPECIAL WASTES

	Surrogate Chemical	UCD Category	Waste Quantity (t/a)
Class 9:	Aqueous Solution with Organics		
	Formaldehyde	225	3000
	Toluene	227	1000
Class 12:	Pesticide and Herbicide Waste		
	Petroleum distillates	241	300
Class 13:	PCB Wastes		
	PCB	242	80
Class 14:	Clean-up Residuals		
	Toluene	521	1400
	Petroleum distillates	531	1400
	TOTAL		59 410

Source: OWMC (1984)

where Q = emission rate for species i, g/s

Kg = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure for species i, Pa

M_i = molecular weight of species i, g/mol

R = gas constant, $(Pa.m^3)/(mol.K)$

T = temperature, K

The gas phase mass transfer coefficient was estimated by a method developed by Mackay and Matsugu (1973) which is explained in detail in Appendix C of this report.

$$Kg = 0.0048\ (U)^{0.78}\ (d)^{-0.11}\ (Sc)^{-0.67}$$

where U = wind speed, m/s

d = diameter of pool, m

Sc = Schmidt number, dimensionless

REAGENTS AND ADDITIVES

The reagents and additives for comparison of relative risks were selected based on the potential for the commodities to cause releases to the air as a result of volatilization of organic chemicals or emissions of particulates during a spill event. The reagents and additives used in the physical/chemical treatment and incineration processes are shown on Table E-2. Those reagents and additives determined to be potentially significant contributors to air emissions in the event of a spill were cement, hydrated lime, activated carbon, and calcium oxide. Caustic soda was not selected

TABLE E-2

REAGENTS AND ADDITIVES

Chemical/Reagent	Form	Quantity (t/a)[1]
Physical Chemical Treatment Plant		
Cement	powder	52 700 for total solidification
Sodium silicate	liquid	21 700
Caustic soda	50% liquid	640
Hydrated lime	solid	7400
Activated carbon	solid	100
Nitrogen[2]	liquid	8000 m^3/a
Incinerator System		
Caustic soda	50% liquid	5600
Calcium oxide	solid	2380
Nitrogen[2]	liquid	16 000 m^3/a

[1]All quantities based on information contained in Phase 4 Engineering Report prepared by Monenco (1985).

[2]Quantities at standard temperature and pressure.

because it is usually shipped in flakes or beads when in solid form and would not readily be subject to wind dispersal (Environment Canada, 1981). The other reagents and additives which were excluded are liquids that are not expected to contain volatile organic chemicals.

In the event a spill of reagents or additives occurs, solid materials would most likely form a waste pile. The waste pile would be subject to erosion and wind dispersal of particulates. The estimates of release potential were determined based on the following equation for estimating particulate emissions from a waste pile (EPA, 1977):

$$M_v = 0.0045(M_{wp})(X_i)/(PE)^2$$

where M_v = constituent particulate emission rate, kg/d

M_{wp} = mass of pile, kg

X_i = mass fraction of constituent, kg/kg

PE = Thornthwaite's precipitation-evaporation index

A value of 110 was used for PE which reflects climatic conditions in the Niagara region of the Great Lakes (EPA, 1977). In addition, the spill event was assumed to be a spill of 100% of the commodity being transported.

In the absence of LD_{50} values for the chemicals and additives selected for evaluation, a derived LD_{50} was approximated on the basis of the Threshold Limit Value (TLV). The TLV is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which

nearly all workers may be repeatedly exposed without adverse effect. The concentration based TLV was first converted to an equivalent dose based on the following equation:

$$\text{Equivalent Dose (mg/kg)} = \frac{\text{TLV} \times \text{BR} \times t \times \text{CF}}{\text{BW}}$$

where TLV = Threshold Limit Value, mg/m^3

BR = Breathing rate, 29 l/min

t = Exposure time, 60 min

BW = Body weight, 70 kg

CF = Conversion factor

A conversion factor must be applied to consider the fact that the TLV is based on exposure for an 8-hour workday and a 40-hour workweek and the LD_{50} considers a one hour exposure time. A factor of 1000 was then applied to the LD_{50} equivalent based on the relationship between the TLV and the LD_{50} (McNamara, 1976) assuming that the TLV is equivalent to the no effect level (NOEL).

The quantity of wastes used in the calculation of the hazard index are shown on Table E-2. These quantities were based on the ultimate plant throughput of 300 000 t/a. The quantity of cement required for total solidification was chosen as representing the most conservative case, i.e., total solidification, in determining the severity index.

INTER-SITE TRANSFERS

The relative risks of inter-site transfers were assessed based on those waste streams which could potentially be transported between treatment units located on different sites. Those waste streams identified included oily wastes after treatment by emulsion breaking and dissolved air flotation at the physical/chemical treatment plant and transported to the incineration site; and ash from the incineration unit transported to the engineered landfill. Surrogate chemicals were chosen to represent the oily waste streams that would be sent to the treatment plant (see Appendix B). The commodities selected to represent inter-site transfers and their quantities are presented in Table E-3.

The release potential for the surrogate chemicals was estimated using the equation presented for volatilization of organic chemicals. The release potential for the ash was estimated using the equation presented for emissions of particulates for waste piles.

Toxicity for 1,1,1-trichloroethane and trichloroethylene was based on the LD_{50}. An LD_{50} was unavailable for incinerator ash, therefore, an equivalent dose was estimated from the TLV using the methodology presented for reagents and additives.

TABLE E-3

INTER - SITE TRANSFERS

Waste	Quantity (t/a)
Oily Wastes Trichloroethylene Benzene	8000
Ash	13 000[1]
TOTAL	23 600

[1]Quantity based on information contained in Phase 4A Engineering Report (Monenco, 1985)

E.3 RESULTS AND ANALYSIS

The results of the hazard index are shown below:

Commodity	Overall Severity Index	Frequency Index (t/a)
Special Wastes	7.43×10^{-2}	59 410
Reagents and Additives	8.54×10^{-8}	62 580
Inter-site Transfers	2.06×10^{-6}	21 000

It should be noted that the indices presented above are relative measures of severity and frequency only and do not have any absolute significance. The indices show that the relative risks of the severity of special wastes transported to the facility, reagents and additives transported to the facility, and inter-site transfers are on the order of of 36 000:0.04:1. In

addition, the ratio of relative frequency of spills for the three commodity groups is determined to be on the order of 2.8:3:1. Although a spill of reagents and additives can occur at approximately the same frequency as a spill of special wastes, the relative severity of the risks posed by reagents and additives is much less significant in comparison to special wastes. Thus, it can be concluded that the risks associated with transportation of special wastes to the facility are much greater than for the transportation of other commodities.

Since these risks were based on average weather conditions in Southern Ontario, consideration was given to how different conditions might influence the relative risks. In particular, lower temperatures and higher wind speeds would tend to decrease the emission rates for volatile organic chemicals (eg., special wastes) relative to particulates (eg., reagents and additives). For example, the emission rate for a volatile organic chemical at 0°C will be approximately 1.8 times lower than at 10°C. In addition, wind speed influences the emission rate for volatiles by its effect on the mass transfer coefficient, Kg. Higher wind speeds will increase the mass transfer coefficient and thus the emission rate. If the wind speed were changed from 3 m/s to 1 m/s, the emission rate would be reduced by a factor of 2.4. The combined effect of a 10°C decrease in temperature and reduction in wind speed from 3 m/s to 1 m/s would be to reduce the overall severity index for special wastes to 1.77×10^{-2}. It is thus apparent that changes in temperature and wind speed will not have a significant effect on the ratio of relative risks.

REFERENCES

Canviro Consultants, Ltd. 1985. Market Assessment Program: Survey of 1000 Industrial Establishments.. Prepared for Ontario Waste Management Corporation.

Environment Canada. 1981. Technical Information for Problem Spills - Sodium Hydroxide. December.

Franklin Associates, Ltd. 1983. Revised Draft Report on Waste Oil Management and Composition. USEPA Office of Solid Waste.

Mackay D., and Matsugu, R.S. 1973. Evaporation Rates of Liquid Hydrocarbon Spills on Land and Water. Canadian Journal of Chemical Engineering. 51:434, August.

McNamara, B.P. 1976. Concepts in Health Evaluation of Commercial and Industrial Chemicals. In Advances in Modern Toxicology, Volume 1, Part 1: New Concepts in Safety Evaluation. Hemisphere Publishing Co. Washington.

Monenco. 1985. Site Selection Process. Phase 4A: Selection of a Preferred Site(s): Engineering. Prepared for Ontario Waste Management.

Ontario Waste Management Corporation. 1982. Facilities Development Process. Phase 1 Report. Prepared for Ontario Waste Management Corporation. September

Ontario Waste Management Corporation. 1984. Waste Profile. November.

Stiver, W. and Mackay, D. 1982. Spill Technology Newsletter, January/February 1982. As cited in Technical Information for Problem Spills. Draft Introduction Manual.

United States Environmental Protection Agency. 1977. Compilation of Air Pollutant Emission Factors. Third Edition, August.